"STUDY OF CORONARY ARTERY DISEASE IN WOMEN"

– CLINICAL PROFILE AND RISK FACTORS

Dissertation submitted to

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M.D. – General Medicine – Branch I

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The Tamilnadu Dr.M.G.R. Medical University

Chennai

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CERTIFICATE

Certified that the dissertation titled **" STUDY OF CORONARY ARTERY DISEASE IN WOMEN" – CLINICAL PROFILE AND RISK FACTORS"** is a bonafide work done by **Dr.T.CHAKRAVARTHI**., under my guidance and supervision, in partial fulfillment of regulations of The Tamilnadu Dr.M.G.R.Medical University for the award of M.D Degree Branch I, (General Medicine) during the academic period from 2013 to 2016.

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DECLARATION

I solemnly declare that the dissertation titled **"STUDY OF CORONARY ARTERY DISEASE IN WOMEN" – CLINICAL PROFILE AND RISK FACTORS"** was done by me at K.A.P.V.Government Medical College, Tiruchirappalli under the guidance and supervision of Prof. **Dr.N.K.SENTHILNATHAN.M.D.,** The dissertation is submitted to the Tamil Nadu Dr.M.G.R.Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree in General Medicine.

Place : Tiruchirappalli
Date : **Dr.T.Chakravarthi.**

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CONTENTS

INTRODUCTION

INTRODUCTION

Cardiovascular disease remains the leading cause of death in women regardless of race or ethnicity. More women than men have died yearly from cardiovascular disease since 1984 in United States. It accounts for 1 in 3 women death. Mortality rates for CAD decreases for both men and women and rate of decrease is less in women than men. A greater proportion of women of 52 % and men of 42 % with ACS died of sudden death before they reaching the hospital[1].

The world wide INTERHEART study has revealed that women develop MI 10 years later than men, but mortality among women is greater.

Cardiovascular mortality has decreased in women similar to men since 1980s. The importance of Coronary Artery Disease and its prevention in women is receiving increased physician attention[234]. Exploration of sex differences also increased. Evidence based guidelines has been updated with expert panel review for prevention of CAD in women.

Death among women is higher than men due to CAD. There was substantial ethnic variation among responses of women in 2005 survey.

CAD risk in Indian Women:

Among Asian men, half of the all MI occur under the age of 50 years and 25 % under the age 40. Excess burden of premature CAD in Asian is due to genetic susceptibility which is mediated by elevated level of Lipoprotein-a (LPa) and smaller caliber of the coronary arteries.

The CAD mortality among Indians is greater among women than men. Three vessel disease on angiography is seen in one third of premenopausal women. The CAD mortality in women between 45 -64 years of age is double than in whites.

In Singapore, CAD mortality among Asian Indian women 30-39 years of age is 8 fold higher than Chinese women of same age.

Angina pectoris due to reversible myocardial ischemia is caused by obstructive CAD that limits blood flow during myocardial oxygen demand. This syndrome will not affect women until they became elderly with the exception of diabetic women.

CAD in India Statistics:

12 % in urban, 4 times more than USA, 6 times more than European, 20 times more than Japanese[6].

- 10 years earlier 53 Vs 63
- Virulent Progression

- Rural also increasing
- 50 % MI occur below 50 years
- Death due to MI is 2 -3 times greater
- 90 patients die of CVD every hour in India

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Cardiovascular disease is the leading cause of death among women. This accounts for more death than from stroke, lung cancer, COPD and breast cancer combined[7]. About half of these deaths results from coronary heart disease. Mortality rate decreases if modifiable risk factors like hypertension, cholesterol, smoking, physical inactivity are modified.

Mortality rate of STEMI among women is higher than men. The symptoms of ACS are atypical like epigastric discomfort, giddiness, and tiredness and also they present late to the hospital.

The first presentation of CAD among women is 10 years later than among men and most commonly after menopause. The onset of CAD is earlier in developing countries. Mortality rate is higher among women than men in both developed and developing countries.

CAD :

Acute coronary syndrome is a term representing a common end result, acute myocardial ischemia. Acute ischemia is usually, but not always caused by atherosclerotic plaque rupture, fissuring, erosion or a combination with superimposed intracoronary thrombosis and is associated with an

increased risk of cardiac death and myonecrosis. It includes STEMI, NSTEMI, and unstable Angina .

ST-segment elevation myocardial Infarction (STEMI) represents the most lethal from of ACS in which totally occluding thrombus results in total cessation of coronary blood flow in the territory of occluded artery and results in ST elevation in ECG.

STEMI :

Stages of STEMI :

1. Early hyperacute phase - within hours
2. Fully Evolved acute phase - within days
3. Fully Evolved subacute phase - within weeks
4. Chronic stabilized phase - within months

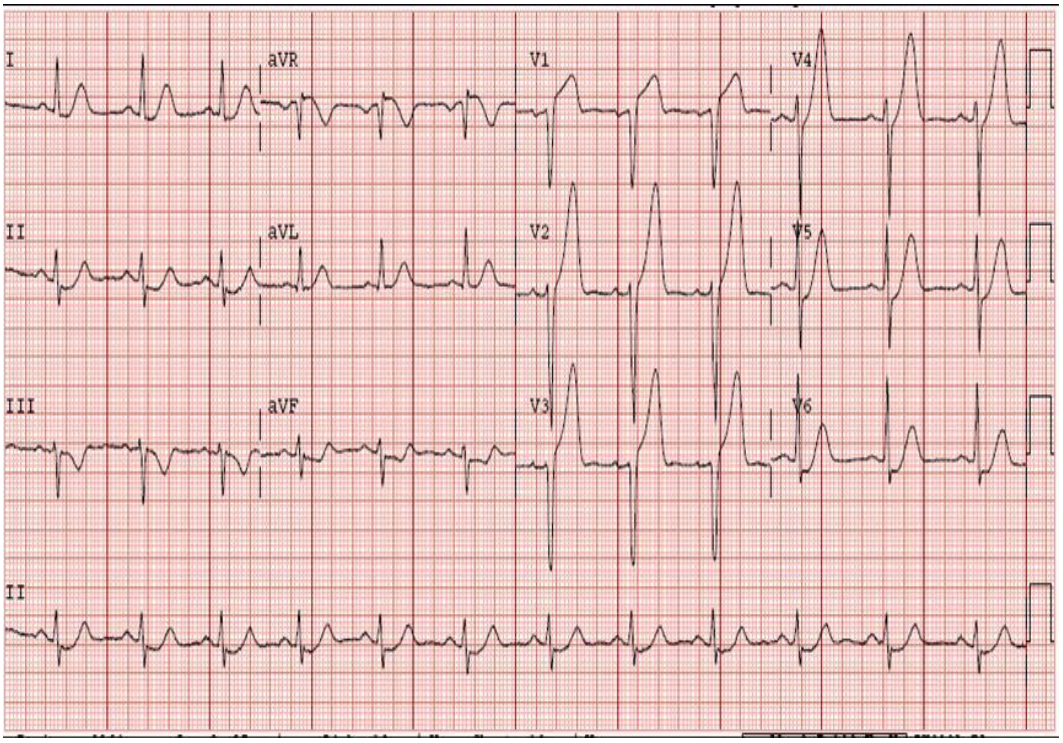
The different modalities of management are planned according to the time of infarction and so it is essential to not only diagonise AMI, but also to identify the stage of AMI.

Early Hyperacute phase :

(Within minutes of coronary occlusion)

This is the most crucial phase of AMI which everyone should identify and treat the patient or refer the patient without delay.

Early, efficient, management of this phase, not only saves many patient lives but also saves significant portion of myocardian from permanent necrosis.



The ECG changes of this phase :

1. Slope elevation of ST segment
2. ST segment elevation with concavity upwards with upright T wave.
3. Reciprocal Depression.

1. Slope elevation of ST segment :

Majority of AMI start as subepicardial injury producing ST segment elevation.

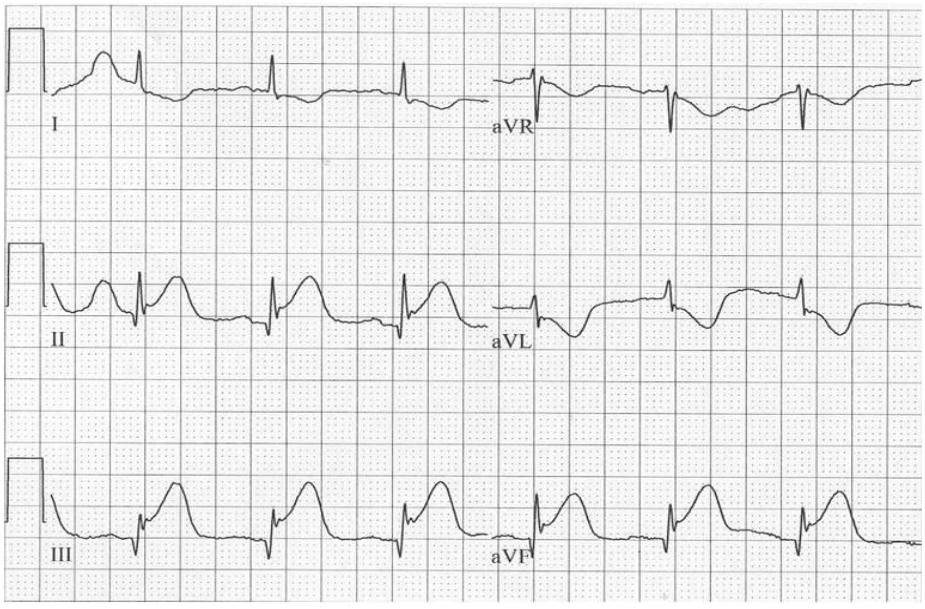
The earliest change is straightening or ironing out of the gentle concavity of ST segment that is usually present between QRS and T wave.

2. ST segment elevation with concavity upwards with upright T wave:

ST segment elevation with concavity upwards with upright T wave which of course is the classical subepicardial injury. In these phases still R wave is present indicating that myocardium is still viable and not necrosed.

3. Reciprocal Depression :

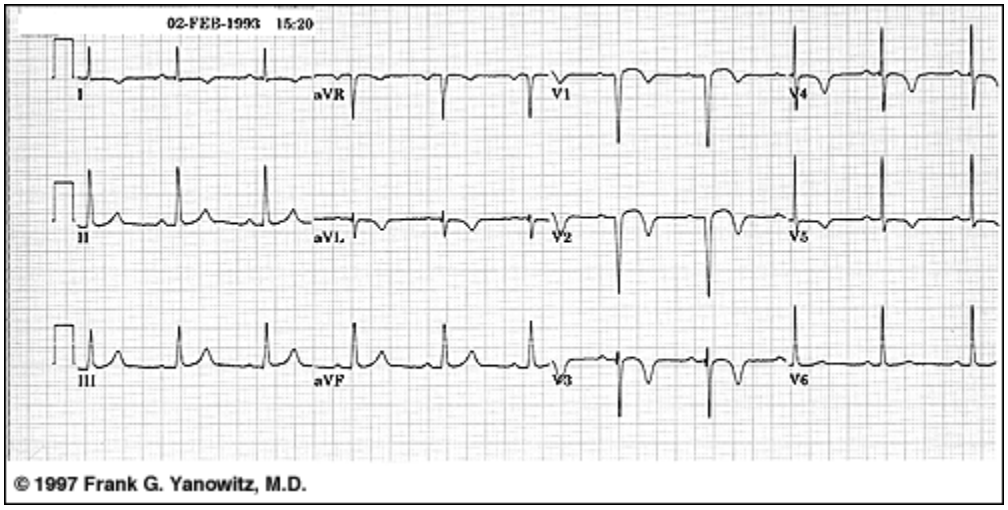
In IWMI, (STEMI) produces ST elevation in inferior leads. It produces reciprocal depression in lateral leads.



Fully Evolved Acute phase :

(within days of coronary occlusion)

In this phase, the portion of myocardium has been already necrosed and there is development of pathological Q waves which represents necrosis and loss of R wave indicating loss of healthy tissue. Efficient management of early hyperacute phase will prevent this.

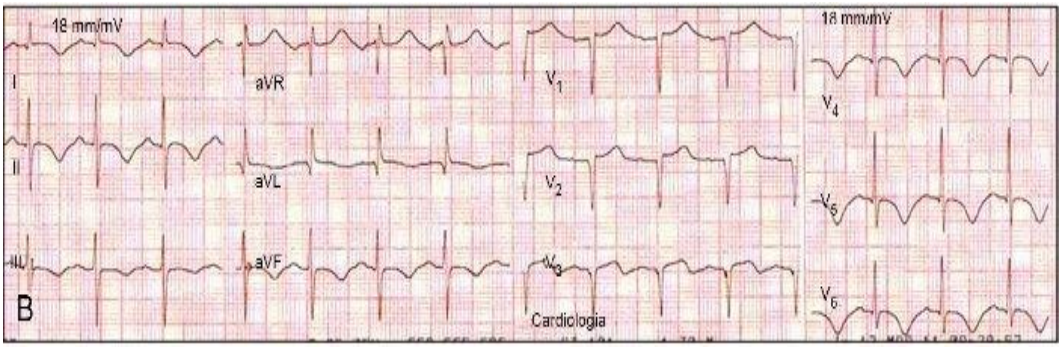


Fully evolved subacute phase:

(within weeks of coronary occlusion)

In this phase, the injury settles down. So the ECG shows pathological Q wave and symmetrical T wave inversion. This phase represents stabilization of acute phase and the patient is usually discharged from the hospital at this stage.

This phase is usually managed with nitrates, aspirin, beta blockers, ACE inhibitors according to LV function. Evaluation of risk stratification to decide about drug therapy or revascularization is also done at this stage.

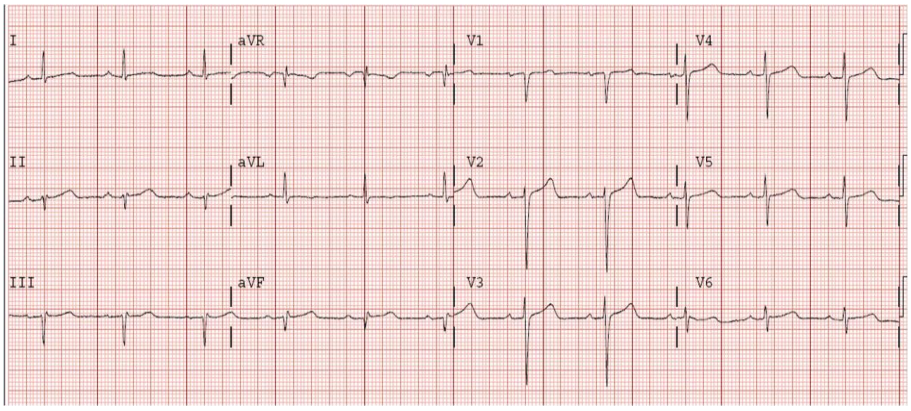


Chronic stabilized phase :

(Within months of coronary occlusion)

In this phase, the ECG shows only pathological Q wave representing the permanently necrosed myocardium. There is no injury or ischaemia. So there is no ST elevation or T wave inversion.

In this phase we should aim to prevent another AMI by means of strict life style modification and drugs like beta blockers and aspirin.

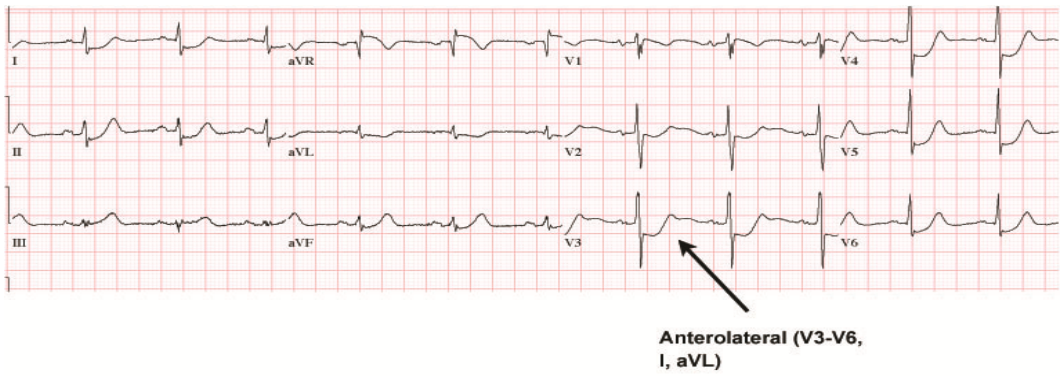


NSTEMI and Unstable Angina:

The diagnosis of acute MI is defined by world Health Organization as having 2 of 3 following Criteria:

1. typical ischemic chest pain
2. typical ECG pattern including Q waves
3. typical rise and fall in serum markers of myocardial injury. Usually CK-MB.

If the patient did not have ST elevation or Q waves and CK-MB was elevated, patient is having NSTEMI. Those patients with chest pain and without evidence of ST segment elevation or Q waves and negative CK-MB level were thought to have unstable Angina.

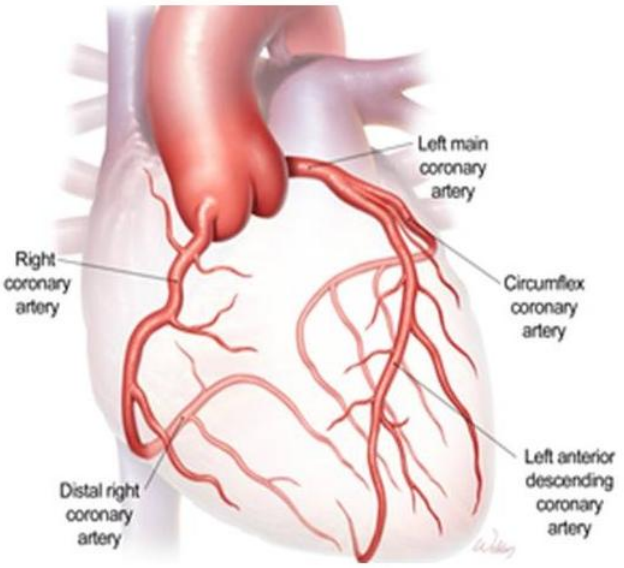


Anatomy of Coronary Circulation :

The Coronaries are the branches from the Right Coronary artery which runs in the right AV groove and divided into posterior descending

artery and posterior left ventricle branches. The artery that give rise to posterior descending branch is the dominant coronary artery.

The left main trunk is 1 cm to 4 cm in length and divides into left anterior descending artery and left circumflex artery. The LAD supplies the Ventricular septum and anterolateral wall. The LCX supplies the Lateral wall of LV and LA.



CAD risk factors and risk factor Modification :

The classic risk factors for CAD are divided into those that are potentially modifiable (like diabetes, hypertension, hyperlipidemia, cigarette smoking, obesity and sedentary life style) and those that are not modifiable (age and family history)

Unmodifiable risk factors :

Genetic polymorphisms that influence the occurrence of cardiovascular events exist between men and women[8], stromelysin member of matrix metalloproteinase enzyme is believed to involve plaque rupture.

Plasminogen activator inhibitor – 1 is also involved in the causation of CAD. The gap junction protein connexin 37, and P^{22} (phox), component of NAD (P) H redox system both involved in MI among men.

Variation in sex hormone and Y chromosome are involved in the pathogenesis of atherosclerosis between women and men. Indeed ex vivo male macrophages responded to androgen with augmentation of 27 genes resulting in increased foam cell formation and LDL degradation.

In female cells did not show response in genes tested. In women plaque composition (more cellular and fibrous tissue) contributes to pathogenesis of atherosclerosis. Estrogen in women produce coronary vasodilation and higher fibrinogen and factor VII levels in women contributes to hemostasis. Men frequently have plaque rupture and women have twice as likely to have plaque erosion as underlying inciting event. With modern development of RNA microarray technology, metabolomics

and proteomic capabilities it is possible to explore sex difference, in genes expression in the development CAD.

Potentially modifiable risk factors:

- Diabetes Mellitus
- Systemic Hypertension
- Hyperlipidemia
- Cigarette smoking
- Obesity
- Sedentary life style

Hypertension :

Hypertension is increasingly common risk factor among Indian population. Women have 15% higher prevalence of hypertension than men. Prevalence of hypertension increases with age in both sexes. Hypertension is higher in women between 45 to 54 years of age than men. < 35 years of age hypertension is more among men than women.

CAD risk is slightly higher among women between 49 to 89 years of age[9]. Unfortunately women are most likely to be undertreated for hypertension than men.

With systolic blood pressure above 180 mm Hg annual incidence of CHD (angina, coronary insufficiency, MI, death) in women older 65 years

was above 30 % but for men older than 65 years it was 50 %[10] Higher diastolic blood pressure produces more CAD events.

Lowering blood pressure decreases the first MI and sudden death. This effect was less dramatic for CAD when compared to stroke.

Angiotensin converting enzyme inhibitors should be used cautiously in women of reproductive age because of teratogenic effects. It produces CV and central nervous system malformations. Other antihypertensive medications didn't increase the risk of congenital abnormalities.

Thiazide diuretics are a preferred first choice in the treatment of hypertension in both men and women and also beneficial for bone health.

Diabetes and the metabolic syndrome :

The prevalence of diabetes is on increasing trend among both men and women[7]. It is expected to double by 2050 across all age and sex groupings. In 2006, women older than 20 years of age represented more than half of the prevalence with known diabetes and accounted for half of the undiagnosed diabetes[7]. Women with prediabetes defined as impaired glucose of 110 to < 126 mg/dl is also increasing trend. Cardiovascular disease is twice as common among women with diabetes as among those without. They are 4 times as likely to be hospitalized and women have a higher risk for most clinical events.

Metabolic syndrome relates closely to insulin resistance and comprises the following risk factors :

- Abdominal Obesity
- Atherogenic lipid Profile

  (Excessive triglycerides or inadequate HDL)
- Blood pressure of 130/85 mnHg or Higher
- FBS of 110mg / dl or greater.

At any given LDL- Cholesterol level metabolic syndrome increases the risk for CAD. After adjustment for age, metabolic syndrome appears to highly prevalent in both sexes with little difference in rates between women and men. Diabetes with premenopausal women carries same risk as with postmenopausal women.

Diabetic patients have higher mortality rates for CAD than nondiabetics. In the past decade, CAD mortality increased to 23 % in diabetic women and decreased of 27 % among nondiabetic women[11].

In diabetic men mortality rate decreased to 13 % and 36 % reduction in nondiabetic men[11]. Sex differences in endothelial function – vasodilation play a pathophysiologic role.

In Framingham heart study, over 30 years CAD occured in 78 % women with diabetes than women without diabeties (38%). Diabetic women

have higher mortality rate after MI and CCF incidence than do diabetic men. Women's increased post MI mortality was associated with hypertension and hyperlipidemia and not with glycemic control.

The relative risk for a CAD event was 2.68 for current diabetic smokers of > 15 Cigarettes daily[12], 1.66 for current diabetic smoker < 15 cigarettes daily. Diabetic women who had not smoked for 10 years had similar risk as of non smoking diabetic women.

Other risk factors control is also essential to decrease overall cardiovascular risk associated with diabetes. Women at risk for developing diabetes include obese women and gestational diabetic women. Even moderate exercise walking (3 hrs / wk) and avoiding weight gain decreases the risk of developing diabetes.

The metabolic syndrome is diagnosed frequently at early age. Tobacco exposure from 12 to 19 years of age dramatically increases the risk of developing metabolic syndrome.

Polycystic ovarian syndrome (PCOS) with raised androgens, low HDL levels and higher triglyceride levels increase CAD risk 10 to 20 % of child bearing women[13] [14]. Regular exercise, avoiding tobacco, treating lipoprotein abnormalities and hypertension is beneficial.

Hyperlipidemia :

47 % of women older than 20 years of age have a total serum cholesterol level > 200 mg/dl and 31.7 % have an LDL cholesterol > 130 mg / dl according to AHA study[7]. Adverse changes in lipid profile accompany menopause, perimenopausal women have triglycerides level rise similar to LDL and Total Cholesterol. Menopause influences HDL cholesterol less dramatically.

HDL cholesterol concentration decline gradually in the 2 years preceeding menopause and levels off after menopause. The postmenopausal increase in CAD risk may result partly from these lipid alterations.

The prevalence of hypercholestrolemia is similar for both men and women. Only 35% women are aware of their diagnosis and 10 % only are under treatment[15].

There are sex differences on the impact of lipids on CV events. HDL is more predictive for women than other lipoproteins. LDL cholesterol increases with age and is more predictive for men. Triglyceride levels may be important in men. Enlarged waist circumference (> 88 Cm) along with elevated triglycerides carry more CV risk for postmenopausal women.

Secondary prevention with drugs for hyperlipidemia decreases CAD events for both men and women.

Lifestyle risk factors:

Overweight is defined as BMI > 25 Kg/m^2. Obesity in women increases cardiovascular mortality. 35 % of women are obese[16]. 65% women are never engaged in vigorous physical activity. only 10 % of women engaged in vigorous activity of 5 days or more per week. Family and occupational stress also contributes CAD risk among women. Smoking among women increases CAD events.

Ethnic and racial differences in obesity occur. Obesity is linked to multiple cardiac risk factors including insulin resistance, diabetes, hypertension and hyperlipidemia and is independently associated with coronary events. The pattern of weight distribution also predict CV events. Apple shape obesity has more risk than pear shape obesity who have weight on the hips and buttocks. A greater waist circumference increases health risk regardless of BMI.

Menopause and hormonal Therapy :

Women with early menopause after gynecologic surgery are facing more CAD risk because of low hormone exposure. Estrogen alone contraindicated in women with uterus because of endometrial cancer.

Psychosocial risk factors :

Both socioeconomic and psychosocial factors affect outcome of CAD[17 18 19]. CAD mortality is greater among those of low socioeconomic status. Level of education, owning a car , income, sex, parental status are taken into account for socio economic status.

Depression is twice more common in women than men and affects outcomes in CAD. Depressive symptoms were common within prior week of CAD in women.

CAD symptoms and diagnosis increased risk of depression more than history of cancer. Depressed young women have more comorbidity and less favorable health. Active treatment of depression reduces the rate of both recurrent MI and death.

Acute and reversible cardiomyopathy has been documented after profound emotional stress.(Tako-tsubo cardiomyopathy) ST elevation MI or dyspnea with severe systolic dysfunction and wall motion abnormalities due to severe depression resolved as little as 5 days or as long as 2 months after treatment.

Global Assessment of risk factors for CAD :

Overall, after adjustment, 9 risk factors accounted for 91 % of population attributable risk for men, 94% among women.

The risk factors were:

- Apolipoprotein B / apolipoprotein A ratio
- Cigarette Smoking
- Hypertension
- Diabetes
- Abdominal obesity
- Psychosocial factors (depression, stress at work or home)
- Financial stress
- One or more life events
- reduced Fruits and vegetables intake exercise and alcohol intake

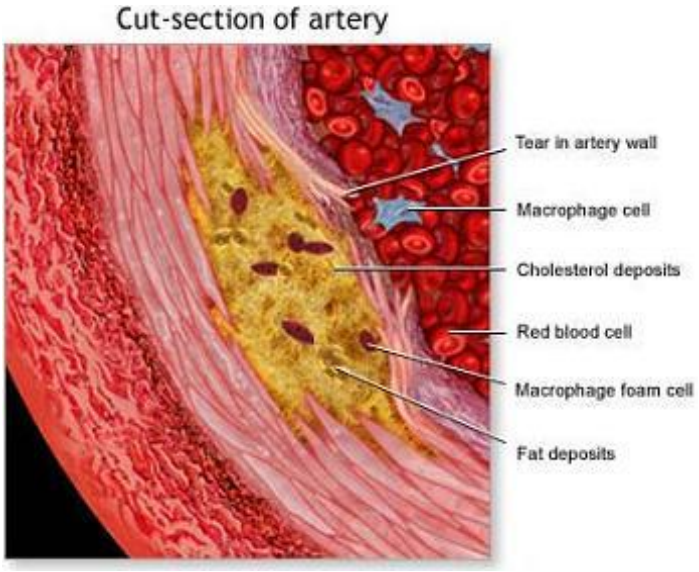
The women with diabetes have more risk than with men with diabetes. Premenopausal women without diabetes are protected from CAD than post menopausal women. Psychological factors also causes more risk for women than men. Healthy lifestyle changes like exercise , moderate alcohol consumption, fruits and vegetable intake protect women from CAD.

Pathophysiology :

Role of Acute plaque rupture

STEMI usually occurs after total occlusion of a coronary artery previously affected by atherosclerosis. Slowly developing coronary artery stenosis do not produce STEMI because of development of rich collateral

network overtime. STEMI occurs when rapidly developing thrombotic total occulsion of coronary artery at site of injury. This injury is aggravated by risks foctors like diabetes, hypertension, cigarette smoking and hyperlipidemia.



Mostly STEMI occurs due to atherosclerotic plaque disrupted. Mural thrombus form at site of plaque rupture and total occlusion of coronary artery occurs. Histologically plaque containing rich lipid core and thin fibrous cap is going to rupture. Collagen, ADP, epinephrine, serotonin, promote platelet activation.

Thromboxane A_2 formed due to activation of platelets produces conformational change in the gp IIb / III a receptor platelet cross-linking and aggregation formed.

Coagulation cascade is activated when tissue factor forms at site of vascular injury. Fluid-phase and clot bound thrombin further activates coagulation cascade. Estrogen can complex with estrogen receptors distributed throughout multiple organ systems. Estrogen may be chronically protective against the development of atheroselerosis in women. Estrogen produces vasodilation.

Rare cases STEMI occurs due to coronary artery occlusion caused by

- Coronary emboli
- Congenital abnormalities
- Coronary Spasm
- Inflammatory diseases

Myocardial damage depends upon[20],

- Area supplied by coronary artery
- Total occlusion or critical occlusion
- Duration of occlusion
- Collateral vessels supply to affected region
- Demand for O_2 of myocardium
    - When total occlusion occurs
- Spontaneous (endogenous) thrombolysis
- Reperfusion of affected area when flow is restored.

STEMI caused by multiple risk factors[21]:

- Unstable Angina
- Smoking
- Hypertension
- Diabetes, Obesity
- Hyperlipidemia
- Sedentary life style

Rare causes of STEMI

- Hypercoagulability
- Collagen vascular disease
- Cocaine abuse
- Intracardiac Thrombi

Clinical Presentation :

50 % of cases have precipitating factor such as vigorous physical exercise, emotional stress, medical or surgical illness. STEMI usually occurs in early morning.

Pain is the most common complaint.

1. Chest pain – deep, Visceral, heavy, squeezing at rest, Typically pain present in the central chest or epigastrium.

2. Sometimes it radiates to arms, neck, jaw, may not go beyond umbilicus, it radiates to occipital area.
3. Associated with sweating, palpitation, nausea, vomiting.

Differential diagnosis of Chest Pain :

- Pulmonary embolism
- Acute Pericarditis – Pain
- Acute aortic dissection – tearing pain beyond umbilicus, back pain
- Costochondritis
- Gastrointestinal disorder

Rarely STEMI presents with

- Painless – Diabetes mellitus, old age
- Sudden onset breathlessness
- Sudden loss of consciousness
- Confusional state
- Weakness
- Arrythmia, giddiness, pulmonary edema

Physical findings :

- Anxious, restless
- Pallor with sweating
- coolness of extremities

- Tachycardia, hypertension (AWMI)
- Bradycardia, hypotension (IWMI)
- Quiet precordium
- S3, S4 – Ventricular dysfunction
    - Decreased intensity of S1
    - Paradoxical splitting of S2
- Midsysolic or late systolic murmur – apex
- Pericardial rub – Transmural STEMI
- Carotid pulse – small volume
- Fever – $38^{o}C$

Lab Diagnosis :

1. ECG
2. Serum Cardiac biomarkers
3. Cardiac imaging
4. Nonspecific indicators of tissue necrosis

ECG :

During intial stage of ACS total occlusion of an epicardial coronary artery produces ST-segment elevation. Most patients then develop Q wave on the ECG following few days. Some patients will not develop Q wave due to rich collateral or not totally occluding thrombus is present. NSTEMI

patient will not develop ST elevation but cardiac biomarkers will be positive.

Cardiac biomarkers :

Troponin T and Troponin I are cardiac specific enzymes which are elevated in myocardial injury. Enzymes > 20 times higher than the upper limit present. Troponin T and I are specific cardiac biomarkers when MI is suspected. Cardiac Troponin are positive in NSTEMI and negative in unstable angina. CPK-MB is more specific then CPK total. Cardiac enzymes are also elevated in cardiac surgery, myocarditis and electrical cardioversion CPK - MB : CK activity > 2.5 suggests myocardial necrosis.

ST Segment elevation MI:

Acute coronary syndrome is one of the most common cause of death in developing world. 50 % ACS death occurs before the patient reaches the hospital. 30 day mortality rate of ACS is 30 % 1 in every 25 patient who recovers from initial hospitalization dies within next one year. Mortality is higher in elderly than in young.

When patient complaints of chest pain with palpitation and sweating, it constitutes Acute coronary syndrome. The 12- lead ECG is important tool of diagnosis of STEMI from NSTEMI.

Unstable Angina and Non STEMI :

Unstable Angina is defined as angina pectoris or equivalent chest discomfort with one of three features,

1. It occurs at rest lasting > 10 minutes;

2. New onset and severe pain within 6 weeks

3. It is distinctly more severe, prolonged, frequent

The diagnosis of NSTEMI is made if unstable angina develops myocardial necrosis with elevated cardiac biomarkers.

Pathophysiology :

UA/NSTEMI is mostly caused by reduction in O_2 supply or increased O_2 demand with atherothrombotic plaque.

Pathologic process :

1. Plaque rupture or erosion with nonocclusion thrombus NSTEMI – Platelet rich thrombus.
2. Dynamic obstruction – Prinzmetal's variant Angina due to coronary vasospasm.
3. Progressive mechanical obstruction-Restenosis following PCI

Inflammation :

Unstable Angina secondary to decreased supply and increased O2 demand . e.g anemia, tachycardia.

Angiography revealed 40 % single vessel disease, 30 % Double vessel disease, 15 % Triple vessel disease and 5 % of UA / NSTEMI has left main artery stenosis.

STEMI has fibrin rich red thrombus and NSTEMI has platelet rich white thrombus.

Risk stratification and prognosis :

10 % patients have 30 days mortality in NSTEMI / UA and 5.15 % have recurrent MI. TIMI trials (thrombolysis in myocardial infarction) includes 7 independent risk factors.

1. Age > 65 years
2. > 3 CAD risk factors
3. Prior stenosis > 50 %
4. ST deviation
5. > 2 Angina events < 24 h
6. While on Aspirin in last 7 days develops UA/NSTEMI
7. Elevated cardiac markers.

Assessment of Reperfusion therapy for STEMI :

Step 1 : Assess time and risk

1.Time since onset of symptoms

2. Risk of STEMI

3. Risk of fibrinolysis

4. Time required for transport for Percutaneous Coronary Intervention

Step 2 : Decide for fibrinolysis or invasive therapy

Fibrinolysis is preferred

1. Early presentation < 3 hr

2.Prolonged transport

3. (Door to ballon) – Door and needle > 1 hr

PCI Preferred :

1. Door to ballon time < 90 minutes

2. Skilled PCI lab is available

3. Cardiogenic shock

4. Killip class > 3

5. Contraindication to fibrinolysis

6. Late presentation > 3 hrs

7. STEMI – Diagnosis is in doubt

Absolute Contraindication to Thrombolysis :

1. Any prior intracranial haemorrhage

2. Cerebral vascular lesion (AVM)

3. Intracranial neoplasm

4. Ischemic stroke within 3 months

5. Suspected aortic dissection

6. Active bleeding or bleeding diathesis

7. Head injury within 3 months

Pathophysiology:

Estrogen activates endothelial Nitric Oxide synthase which inhibits intimal hyperplasia, and smooth muscle migration and promotes antioxidant effects. Estrogen has favourable effects on fibrinogen, plasma viscosity, PAI – I, insulin sensitivity, homocysteine and measures of platelet aggregation. Estrogen influence cardiovascular disease via acting on multiple organ system.

Apolipoprotein gene expression in response to estrogen result in LDL reduction and HDL elevation. Adding progesterone to estrogen replacement therapy produces HDL rise. HRT is associated with increased C-reactive protein which produces inflammation in ACS.

Clinical Presentation :

Most women with ACS present with atypical symptoms like referred pain, in the jaw, neck, shoulder, nausea, vomiting and dyspnea also present in the ACS.

Women mostly present later in the course of symptoms compared with men and most of them have hypertension, tachycardia and heart failure. Unstable angina is more common in women. Women with ACS are older and have strong family h/o of heart disease. They are having comorbidity like diabetes, hypertension. There is no difference in outcome between men and women in ACS.

Atypical symptoms and nondiagnostic ECG findings in women make the ACS diagnosis difficult. Even when cardiac biomarkers are elevated approximately 30% of women have normal coronaries in angiography.

Hormone modulation :

The estrogen replacement and atherosclerosis trial evaluation showed no benefit of HRT on angiographic progression of disease. The selective estrogen receptor modulation carry same risks as HRT including increased incidence of DVT. HRT as whole produce no benefit. Sometimes it produce deep vein thrombosis.

Relative Contraindications:

1. Severe Hypertension > 180 / 110 mmHg
2. h/o ischemic stroke > 3 months
3. Prolonged CPR (> 10 minutes)
4. Recent (3 weeks) internal bleeding

5. Pregnancy

6. Active Peptic Ulcer

7. Use of anticoagulants

8. Non compressible vascular puncture

Hospital Management :

1. Keep vein open with normal saline

2. Vitals monitoring

BP systolic between 100 to 150mmHg

HR between 60 to 100 / mt

RR between 8 to 22 / mt

3. Continuous ECG monitoring for arrhythmia and ST-segment deviation.

4. Diet sips of water until stable.

cholesterol (200 g / day), low fat diet.

5. Bed rest.

6. Oxygen Nasal cannula – 2 liter / min.

7. Medications :

a) sublingual Nitrate 5 mg

IV NTG (Nitroglycerin) for hypertension

b) Aspirin :

Chewable, Nonenteric coated

Aspirin 325mg The maintenance 150 mg OD.

c) Beta blocker (Metoprolol)

d) ACE inhibitor

e) Pain relief : inj morphine 2 -4 mg increment

f) Anxiolytics

g) Daily stool softener

Complications of Acute coronary syndrome :

1. Mechanical Complications

2. Electrical Complications

Mechanical Complications of acute STEMI

1.Cardiogenic shock

2.RV infarction

3.Acute mitral Regurgitation

4.Ventricular septum rupture

5.Free wall rupture

Electrical Complications :

1.Bradyarrythmias

Sinus Bradycardia

Second degree Atrioventricular block

Complete heart block

Bundle Branch block

2.Tachyarrythmias

Ventricular Tachyarrythmias

Supraventricular Arrythmias

Diagnosis of coronary Artery disease in Women :

CAD is often diagnosed with careful clinical history non invasive stress testing aids in the treatment of individuals with intermediate risk for CAD. Unfortunately each noninvasive technique has limitations in women.

Exercise stress testing is noninvasive way to assess CAD risk. Women has lower sensitivity and specificity with exercise stress testing than men due to low ECG voltage. Women have more frequent ST – T wave abnormalities. Negative stress testing reduces the need for catheterization. Stress imaging tests are used for assessing CAD severity.

Nuclear stress perfusion testing with technetium is preferred for assessing CAD severity. Angina symptoms are less predictive of abnormal coronaries in women than men. Early multi detector CT Angiography reveals important sex differences in specific type of coronary lesions.

Management of coronary Artery disease in women :

Asymptomatic Women :

Some patients are truly asymptomatic with respect to CAD. Often they have atypical symptoms undiagnosed as possible CAD. In several studies, more than 25% of MI were not clinically recognised as history of angina was lacking. After 34 years of Framingham follow – up 34 % of women and 26 % of men had MI unidentified. Mortality for women is similar after an unrecognized or recognized MI.

Counseling for asymptomatic women about CAD should include review of common risk factors and symptoms of CAD and advice of healthy lifestyle. Single most preventable risk factor is tobacco smoking. From data in NHANES III (National Health and Nutrition Examination Survey) 90 % CAD events occur among those with atleast one of the following risk factors. Smoking, Blood pressure, Low HDL level, and glucose intolerance. When more than one risk factor are present CAD risk increases.

The first presentation of symptomatic CAD is typically angina in women and MI in men. The prevalence of angina in women and men is similar. Both women and men with new onset CAD benefit from symptom control and secondary prevention. Daly et al reported 3779 men and women diagnosed with stable angina. Anginal symptoms in women are less

predictive of abnormal coronary anatomy than man. Women had less revascularization and more death and nonfatal MI during one year follow up. Secondary prevention should be initiated with the diagnosis of angina including risk factor control and therapy (aspirin and lipid lowering agents) but usually not hormone replacement therapy.

Prevention of CAD :

Tobacco exposure is important coronary artery risk factor for women and men. The greater tobacco exposure in amount and duration is related to higher CAD events. The number of female smoker in India has increased from 5.3 million to 12.2 million in last three decades.

Cigarette smoking has been associated with earlier first CAD event. Middle aged women experiences less symptomatic CAD than men. Increased risk of MI and death related to tobacco use is higher for women than men. Successful tobacco cessation for women as well for men dramatically decreases the risk for further coronary events.

Weight gain with tobacco cessation is an average 7 to 10 lb and is more among women who inhale more than 25 cigarettes per day. To avoid weight gain with tobacco cessation, several interventions recommended. Exercise, careful selection of snacks, pharmacotherapy and physical activity are advised for reducing weight gain.

Nicotine use doubles the rate of successful tobacco cessation Nicotine patch has higher compliance rate than gum, spray and lozenge. Bupropion is also effective in tobacco cessation and minimize weight gain. On stoppage of drug bupropion weight gain will occur.

Bupropion is contraindicated in patients with seizures, head trauma, heavy alcohol consumption because it lowers seizure threshold. It should be avoided in anorexia and bulimia, recent use of monoamine oxidise inhibitor. Varenicline is more effective and safe in women and men with stable CAD.

Black smokers has more trouble in giving up tobacco than white smokers. Black smokers were found to have higher blood levels of nicotine than white smokers. Physicians have a powerful effect on smoking cessation. Activities to reduce weight gain and stress social support are effective for women.

Acute Coronary syndrome :

There are substantial sex differences in the presentation and natural history of acute coronary ischemia. Women with ACS may present with upper abdominal symptoms (Nausea) neck or jaw pain, shortness of breath. Women were older and higher rates of hypertension, diabetes mellitus and pulmonary congestion than men. Women had higher mortality with STEMI and lower mortality compared with men for NSTEMI and unstable angina. >

25 % had nonobstructive disease. Aggressive management of associated risk factors after MI may benefit women.

Intervention for CAD :

Women have more short term complication rates which are related to older age , medical comorbidites and longer duration of symptoms. Common vascular complication like problems at access site, retroperitoneal bleeding and higher blood transfusion rate. CABG surgery more commonly done in men than women. Women were more likely to have emergency surgery and its complications.

Congestive Heart failure :

Women with CHF tend to have preserved systolic function and are older with hypertension. Although use of ICDs have increased among patients with low systolic EF. Sudden death occurs more often in men than women. Arrythmia are more common in women than men. Aggressive management and secondary prevention for documented CAD will improve symptoms.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

- To find out the risk factors of CAD in women in Trichy.
- To study clinical profile of CAD in women in Trichy

MATERIALS AND METHODS

MATERIALS AND METHODS

Materials :

Source of Data

This study was conducted at M.G.M Govt hospital attached to K.A.P.V.Government Medical College, Tiruchirappalli, Tamilnadu in collaboration with the department of Cardiology during the period of September 2014 to August 2015.

Study Design :

A prospective observational

Unicentric study

Study Duration :

12 months

Ethical Committee Approval :

Ethical committee approval obtained from the Institutional Ethical committee.

Inclusion Criteria :

Study were conducted on 80 patient ranging in women aged 38 to 80 years . They were grouped in to two.

Group A - Premenopausal women

Group B - Postmenopausal women

Female patient who are admitted with symptoms and ECG or ECHO evidence of CAD.

All the particulars were inquired by a questionnaire containing their history, personal h/o, family h/o.

Exclusion Criteria: OP patients

Consent :

An informed consent was obtained from all the patients.

Methods of collection of data :

Blood sample collection

Nearly 8 ml of blood was collected in the fasting state and the below mentioned factors have been analysed. Fasting blood sugar, lipid profile, are taken in empty stomach. Hb_1AC, Urine PCR, Urea creatinine were estimated in another sample.

Blood Pressure recording:

BP was recorded in sitting position with sphygmomanometer. Based on Korotkoff sounds BP was recorded. BP at which the sounds started to appear had been taken as systolic value of BP and that when sound disappears had been taken as diastolic pressure. The size of the BP cuff used was 12 X 22 cm.

Calculation of body mass index :

Anthropometric measurements :

Height was recorded with tape to the nearest one centimeter.

Subjects were instructed to stand upright without shoes with their back against the wall, foot together and eyes directed forward.

Weight was measured with weighing machine using spring balance that was kept on firm horizontal surface. The scale was checked on daily basis and calibration was done with known weights. Weight was recorded to the nearest 0.5 Kg.

The formula given below has been used to calculate BMI :

BMI = Weight in KG / (Height in Meters)^2

Analysis of data :

The information collected regarding all the cases were tabulated in a master chart. Data was analysed with help of statistical software tool epidemiological information package (EPI 2002). Using this software range, frequencies, percentages, means, standard deviations, and p values were calculated.

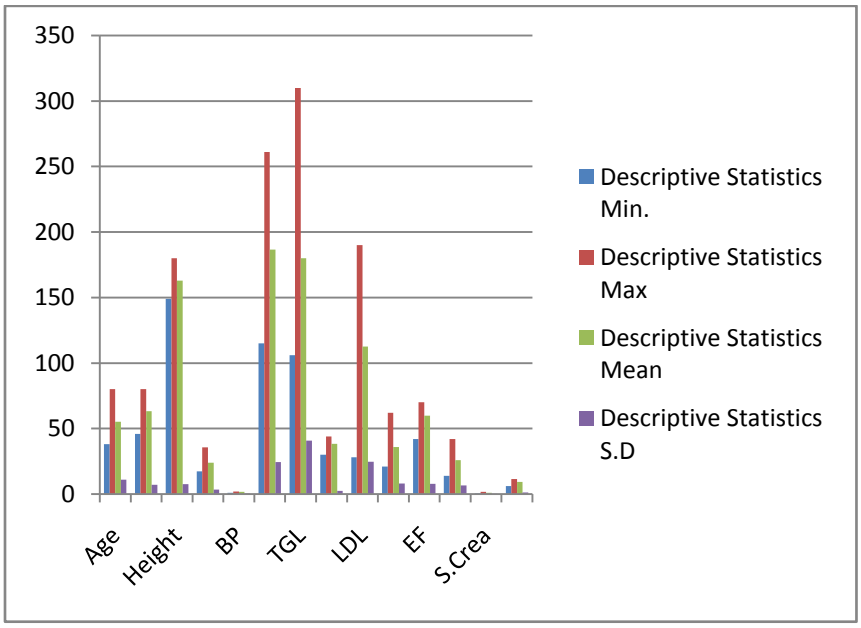
RESULTS

RESULTS

Descriptive Statistics

Item	Min.	Max	Mean	S.D
Age	38	80	55.29	10.994
weight	46	80	63.28	7.144
Height	149	180	162.97	7.567
BMI	17.36	35.56	23.9367	3.33910
BP	110/70	180/110	145/90	4.68
TC	115	261	186.59	24.474
TGL	106	310	179.93	40.873
HDL	30	44	38.45	2.490
LDL	28	190	112.54	24.720
VLDL	21	62	35.99	8.175
EF	42	70	59.77	7.741
Bld.urea	14	42	25.95	6.601
S.Creat	.60	1.60	.9875	.25364
HB	6.00	11.50	9.3363	1.15969
Hb_1AC	4.6	9.5	7.5	1.5

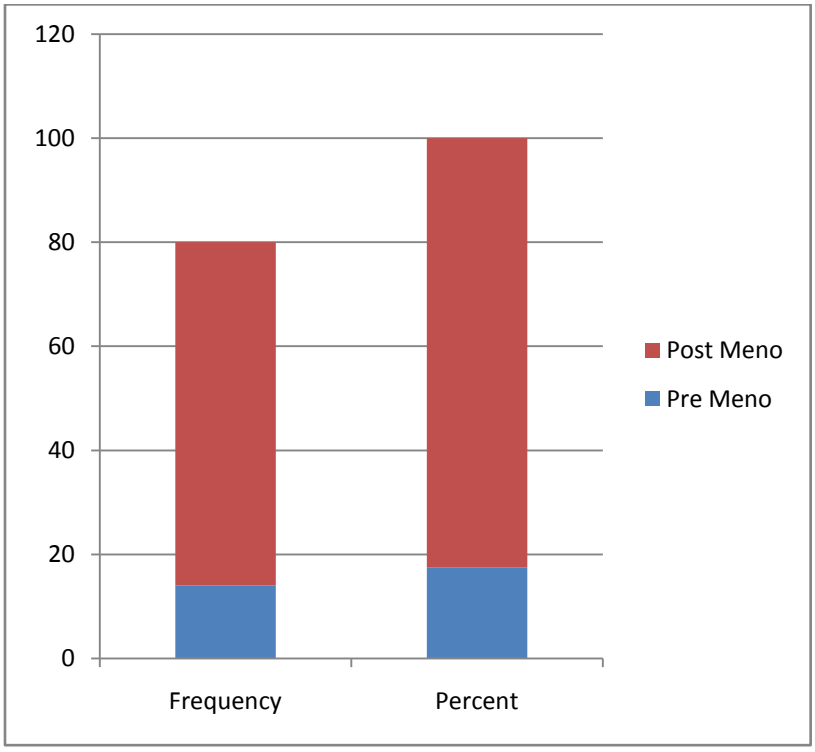
Graph


350
300
250
200
150
100
50
0
Age
Height
BP
TGL
LDL
EF
S.Crea
Descriptive Statistics Min.
Descriptive Statistics Max
Descriptive Statistics Mean
Descriptive Statistics S.D

Sample

Particulars	Frequency	Percent
Pre Meno	14	17.5
Post Meno	66	82.5

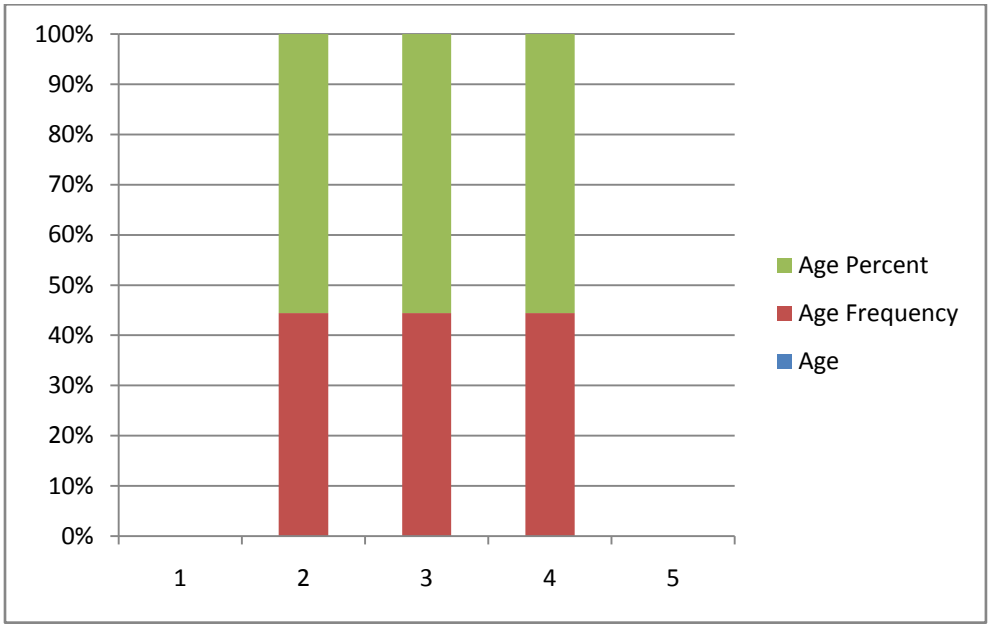
Graph



Age

Particulars	Frequency	Percent
Below 45yrs	14	17.5
46 to 59yrs	41	51.3
60yrs & above	25	31.3

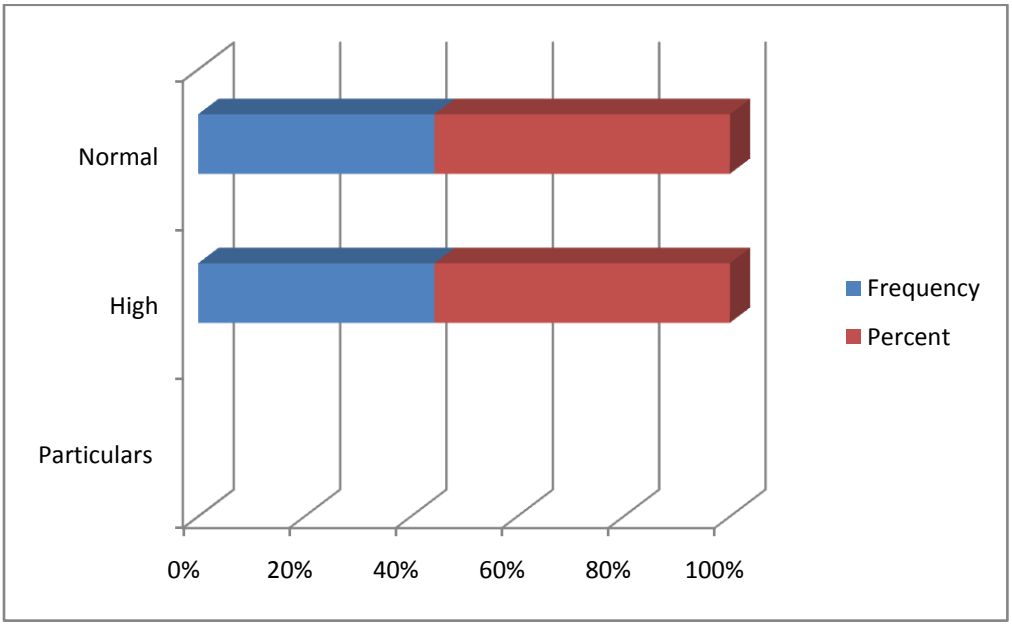
Graph



BP

Particulars	Frequency	Percent
High	30	37.5
Normal	50	62.5

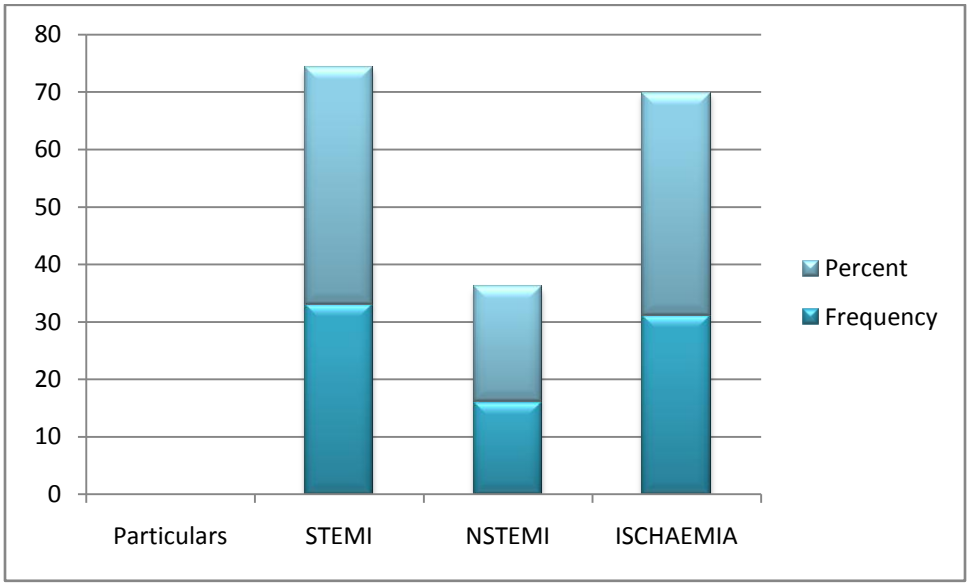
Graph



ECG

Particulars	Frequency	Percent
STEMI	33	41.3
NSTEMI	16	20.0
UNSTABLE ANGINA	31	38.8

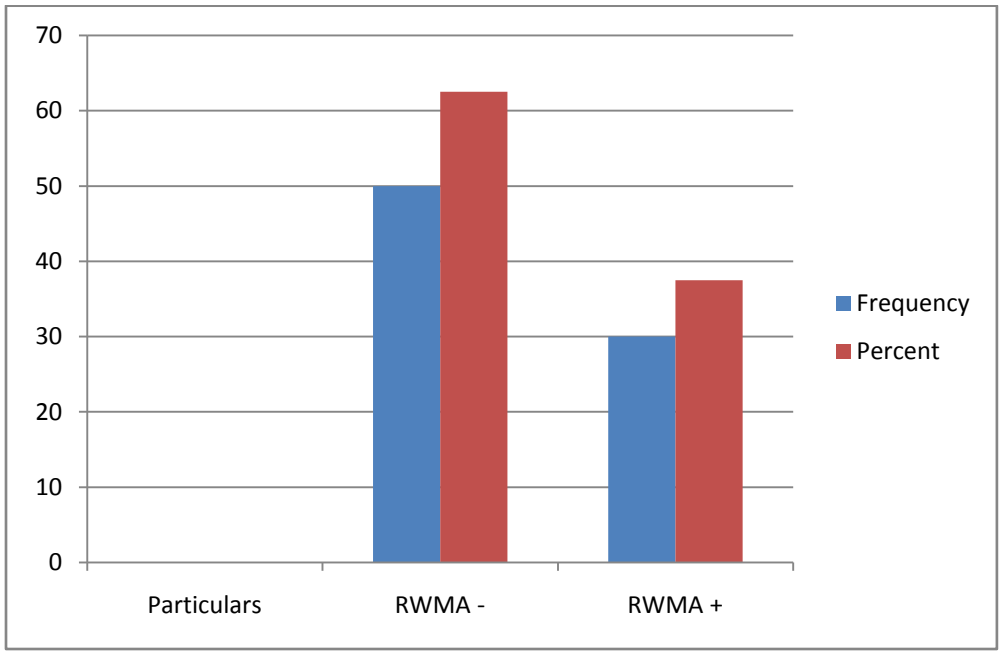
Graph



ECHO

Particulars	Frequency	Percent
RWMA -	50	62.5
RWMA +	30	37.5

Graph

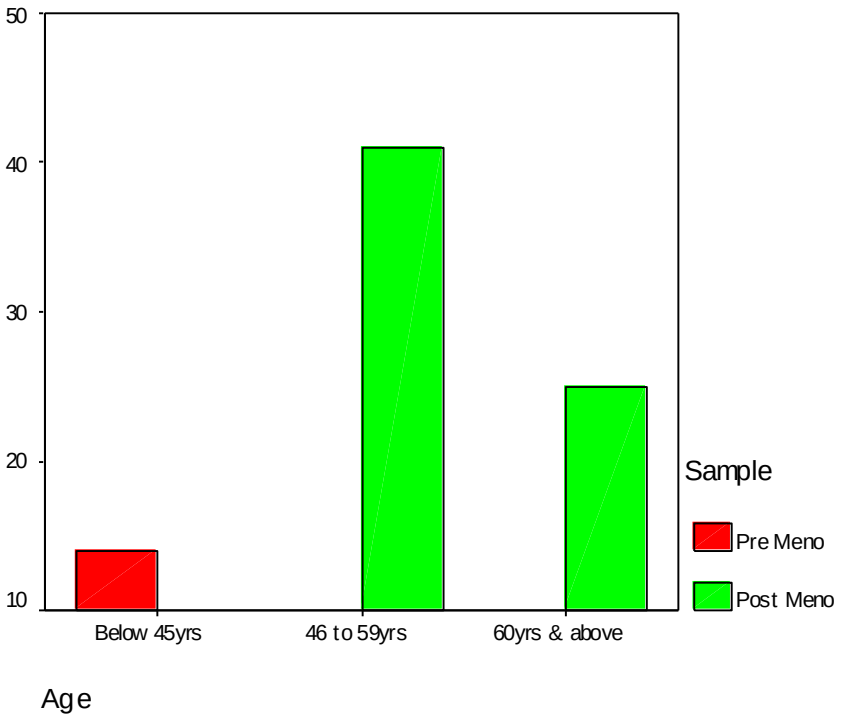


Table

Chi-square test

Age	Pre Meno (n=14)	Post Meno (n=66)	Total (n=80)	Statistical inference
Below 45yrs	14	0	14	X^2=80.000 Df=2 .000<0.05 Significant
46 to 59yrs	0	41	41	
60yrs & above	0	25	25	

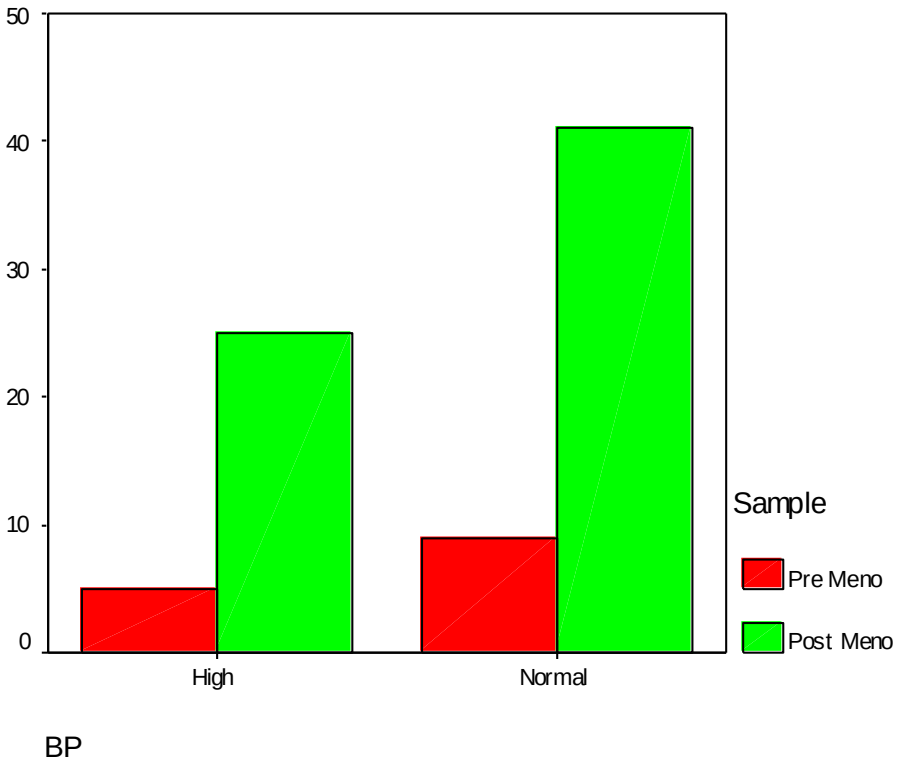
Graph



Chi-square test

BP	Pre Meno (n=14)	Post Meno (n=66)	Total (n=80)	Statistical inference
High	5	25	30	X^2=0.023 Df=1 .879>0.05 Not Significant
Normal	9	41	50	

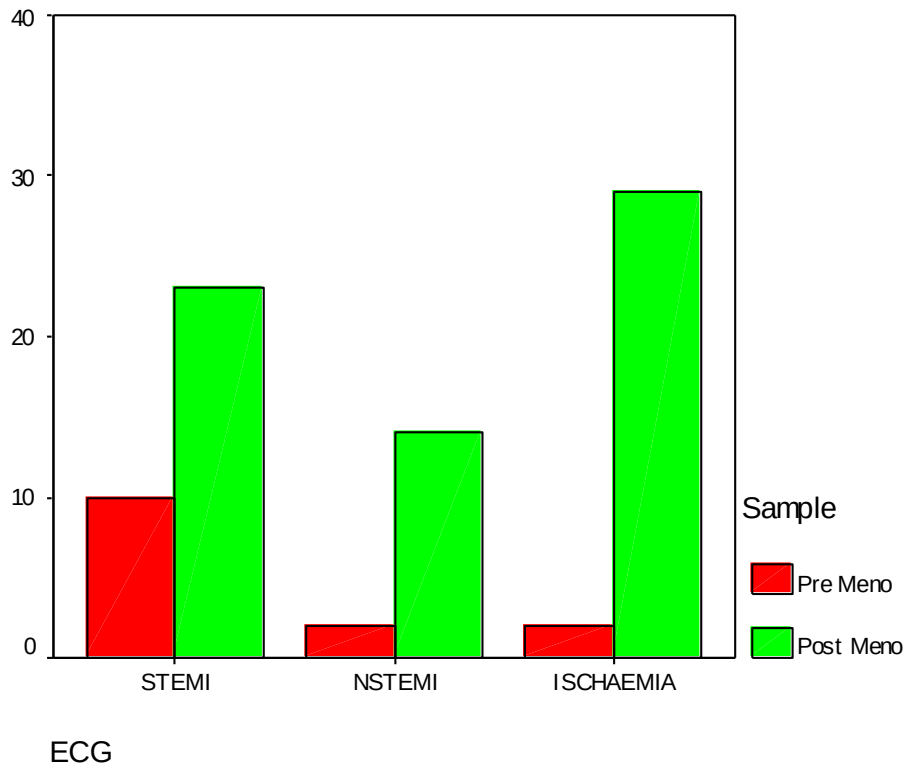
Graph



Chi-square test

ECG	Pre Meno (n=14)	Post Meno (n=66)	Total (n=80)	Statistical inference
STEMI	10	23	33	X^2=6.645 Df=2 .036<0.05 Significant
NSTEMI	2	14	16	
ISCHAEMIA	2	29	31	

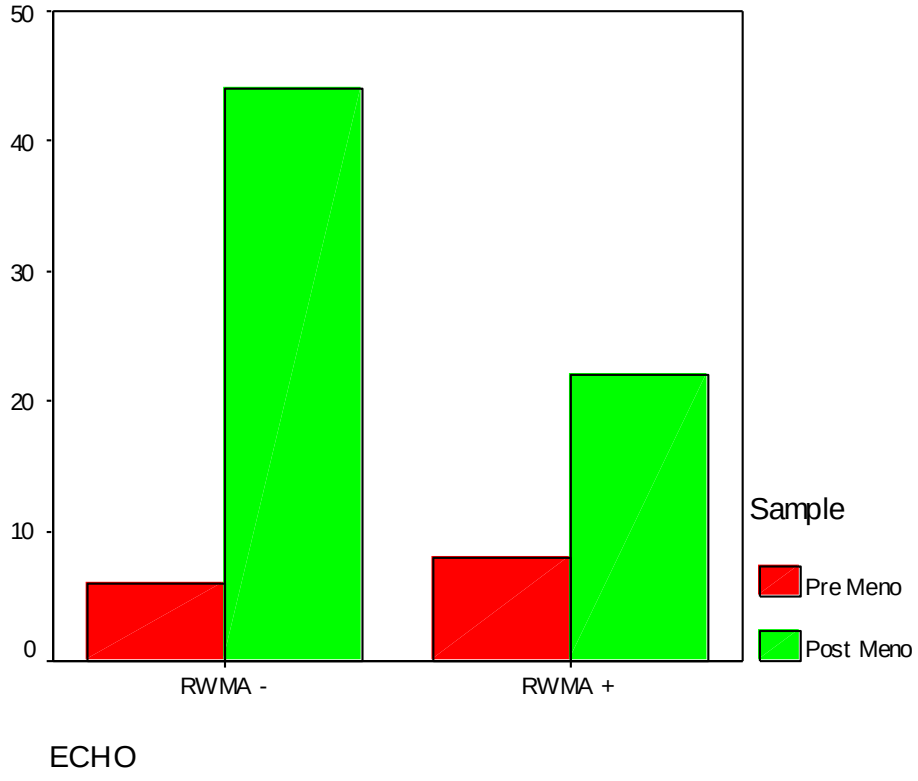
Graph



Chi-square test

ECHO	Pre Meno (n=14)	Post Meno (n=66)	Total (n=80)	Statistical inference
RWMA -	6	44	50	X^2=2.794 Df=1 .095>0.05 Not Significant
RWMA +	8	22	30	

Graph



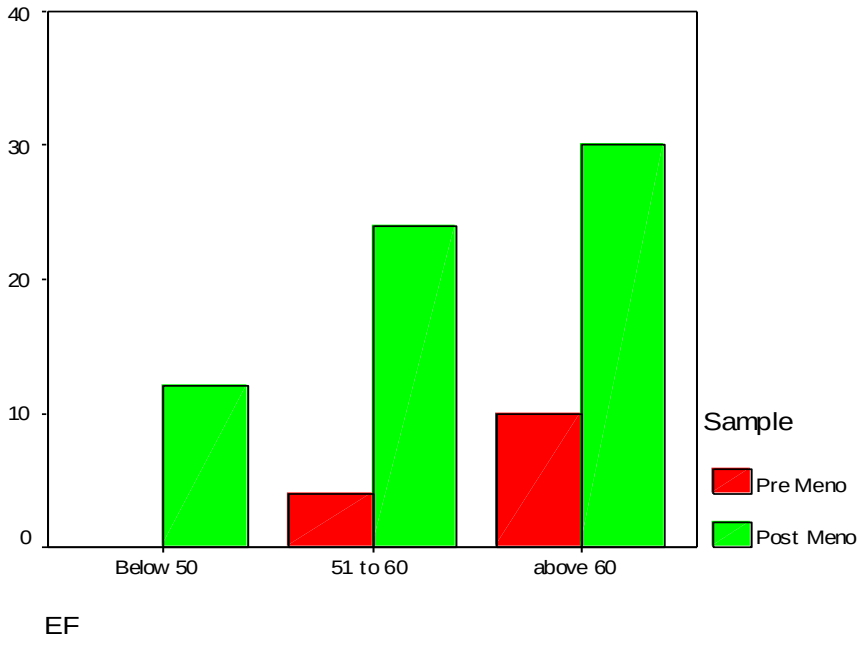
EF

Particulars	Frequency	Percent
Below 50	12	15.0
51 to 60	28	35.0
above 60	40	50.0

Chi-square test

EF	Pre Meno (n=14)	Post Meno (n=66)	Total (n=80)	Statistical inference
Below 50	0	12	12	X^2=4.304 Df=2 .116>0.05 Not Significant
51 to 60	4	24	28	
above 60	10	30	40	

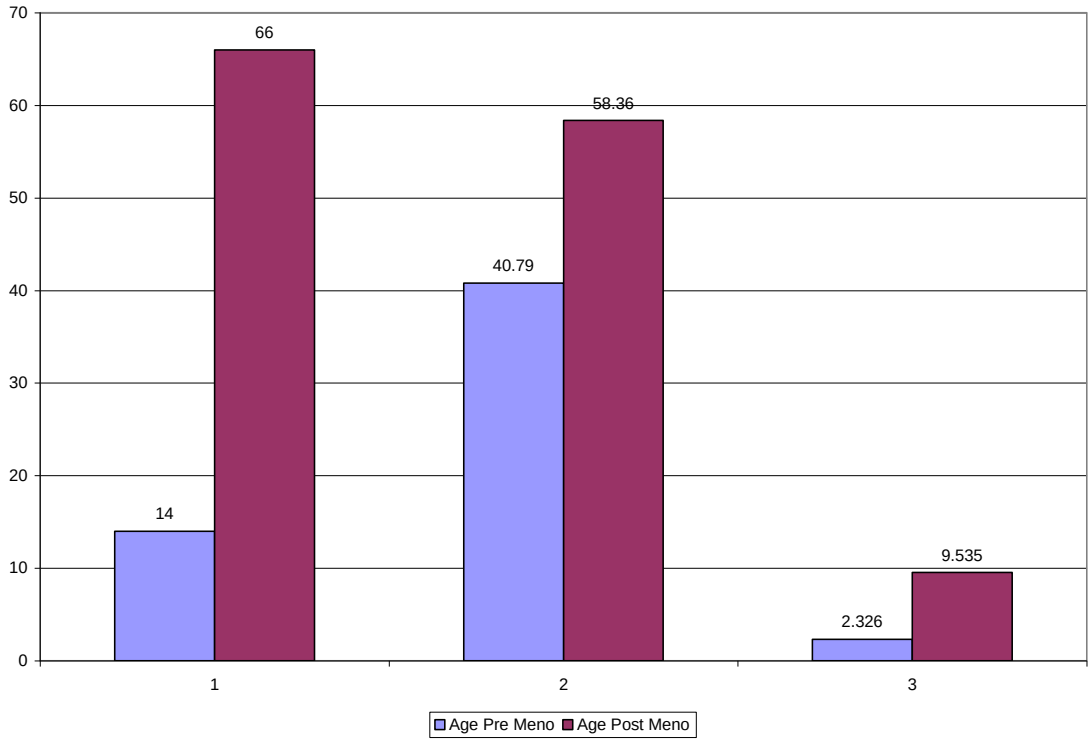
Graph



T-Test

Age	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	40.79	2.326	-6.823	78	.000<0.05
Post Meno (n=66)	58.36	9.535			Significant

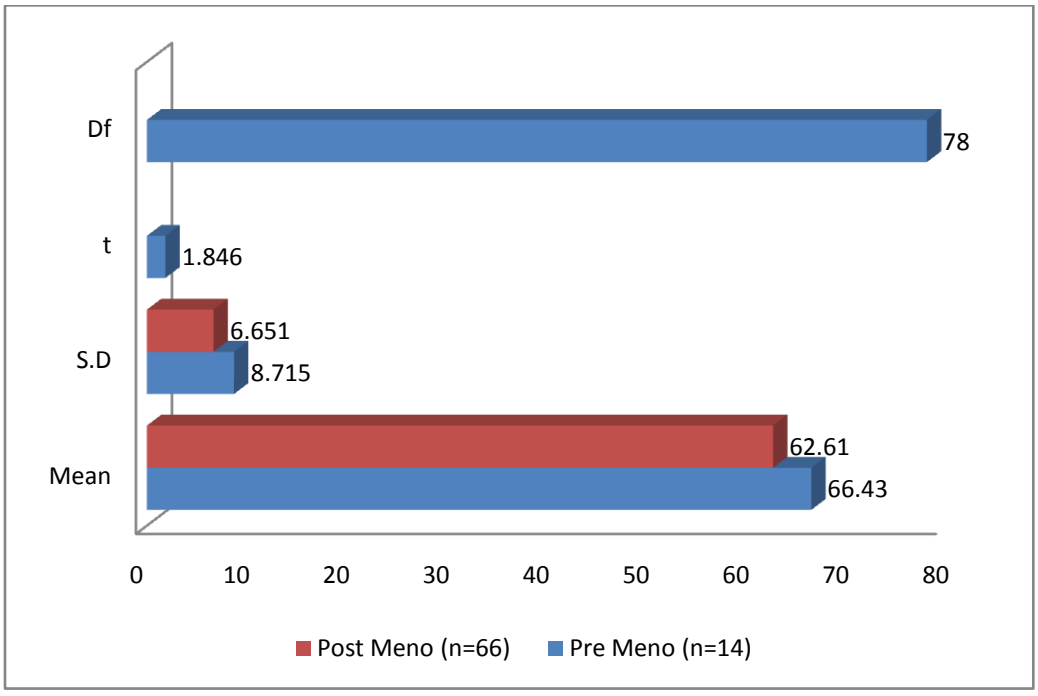
Graph



T-Test

weight	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	66.43	8.715	1.846	78	.069>0.05
Post Meno (n=66)	62.61	6.651			Not Significant

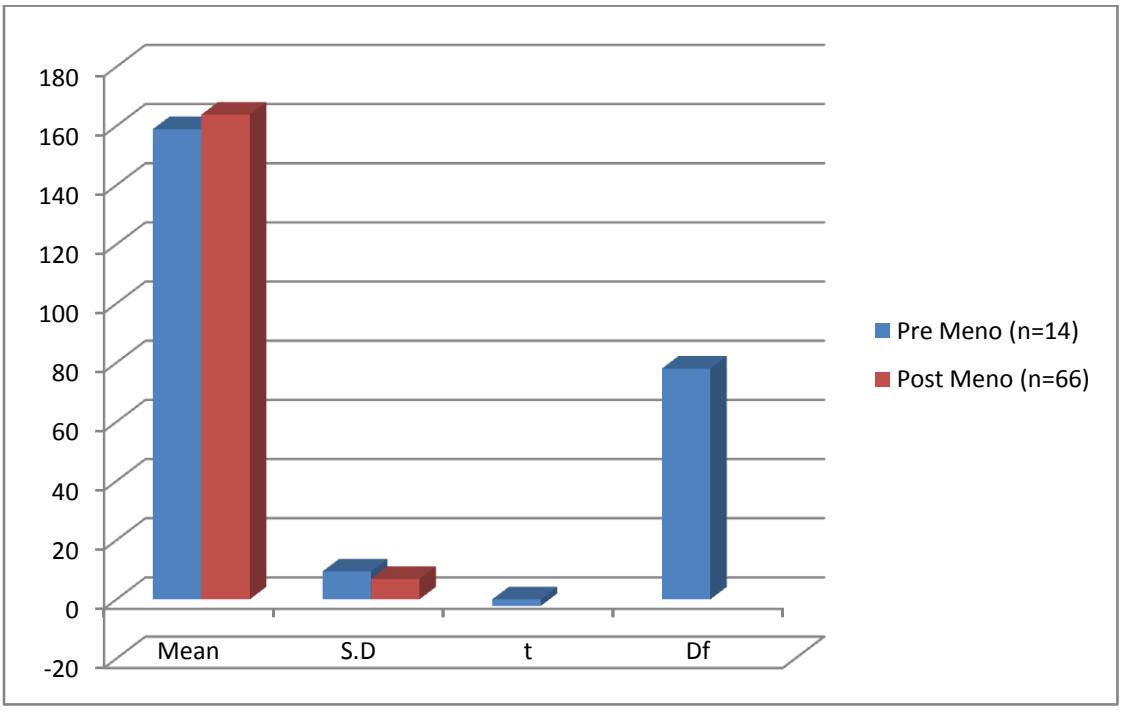
Graph



T-Test

Height	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	158.93	9.442	-2.259	78	.027<0.05
Post Meno (n=66)	163.83	6.892			Significant

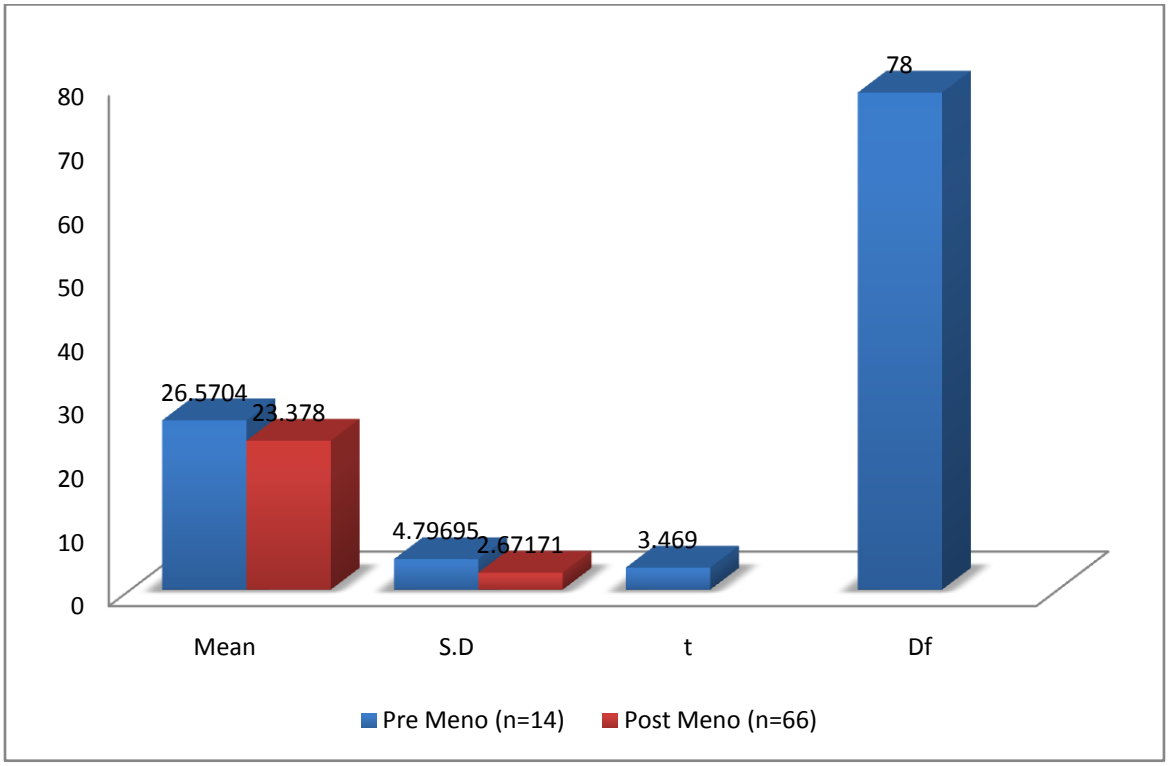
Graph



T-Test

BMI	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	26.5704	4.79695	3.469	78	.001<0.05
Post Meno (n=66)	23.3780	2.67171			Significant

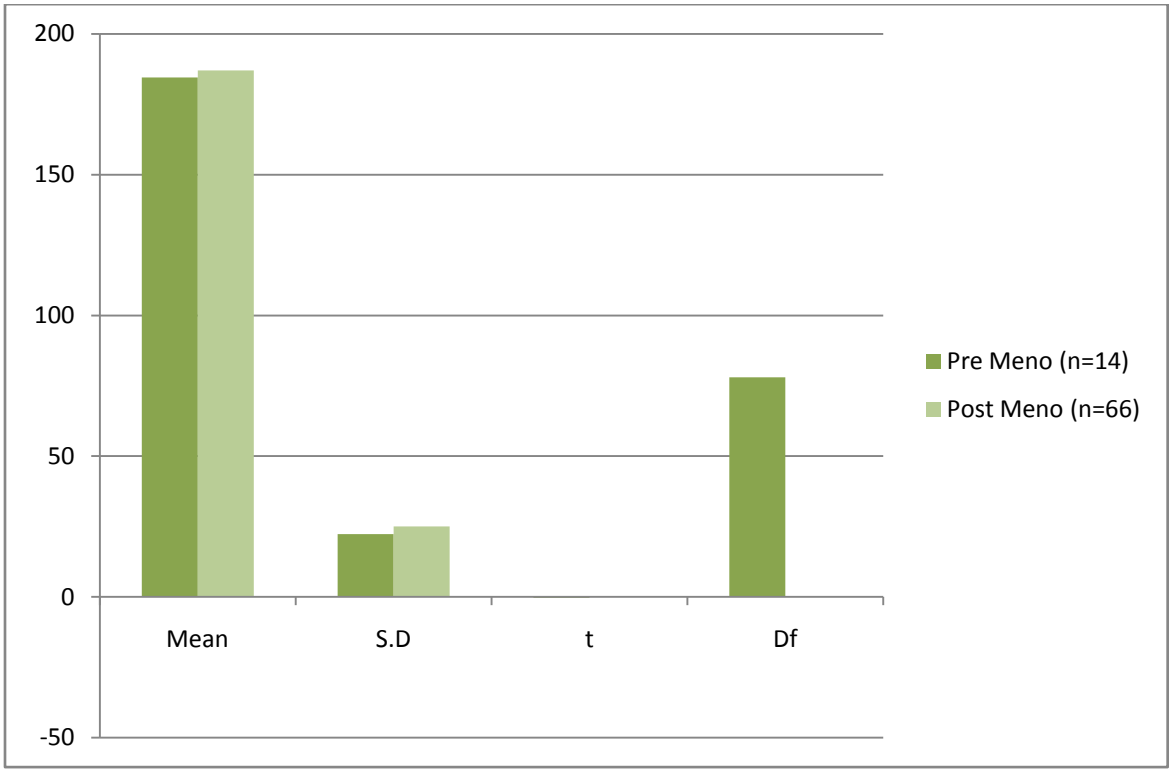
Graph



T-Test

TC	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	184.57	22.301	-.337	78	.737>0.05
Post Meno (n=66)	187.02	25.049			Not Significant

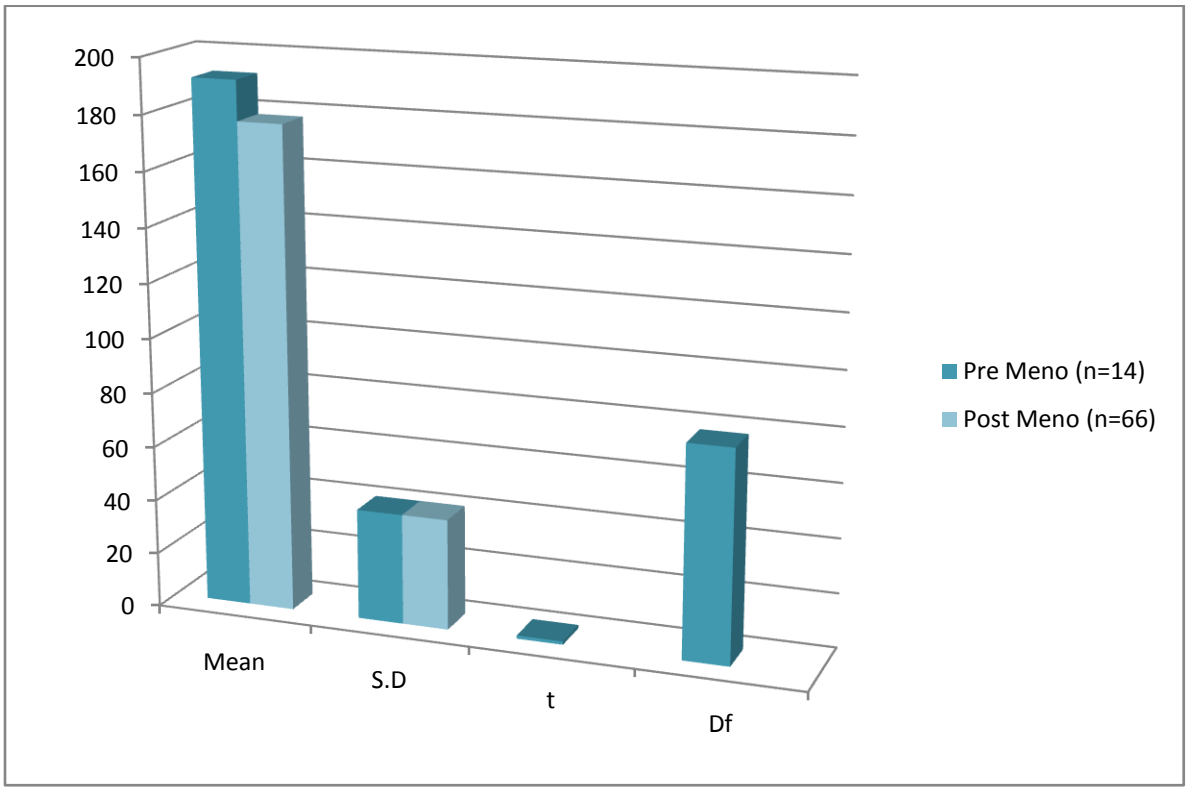
Graph



T-Test

TGL	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	191.86	40.733	1.206	78	.231>0.05
Post Meno (n=66)	177.39	40.761			Not Significant

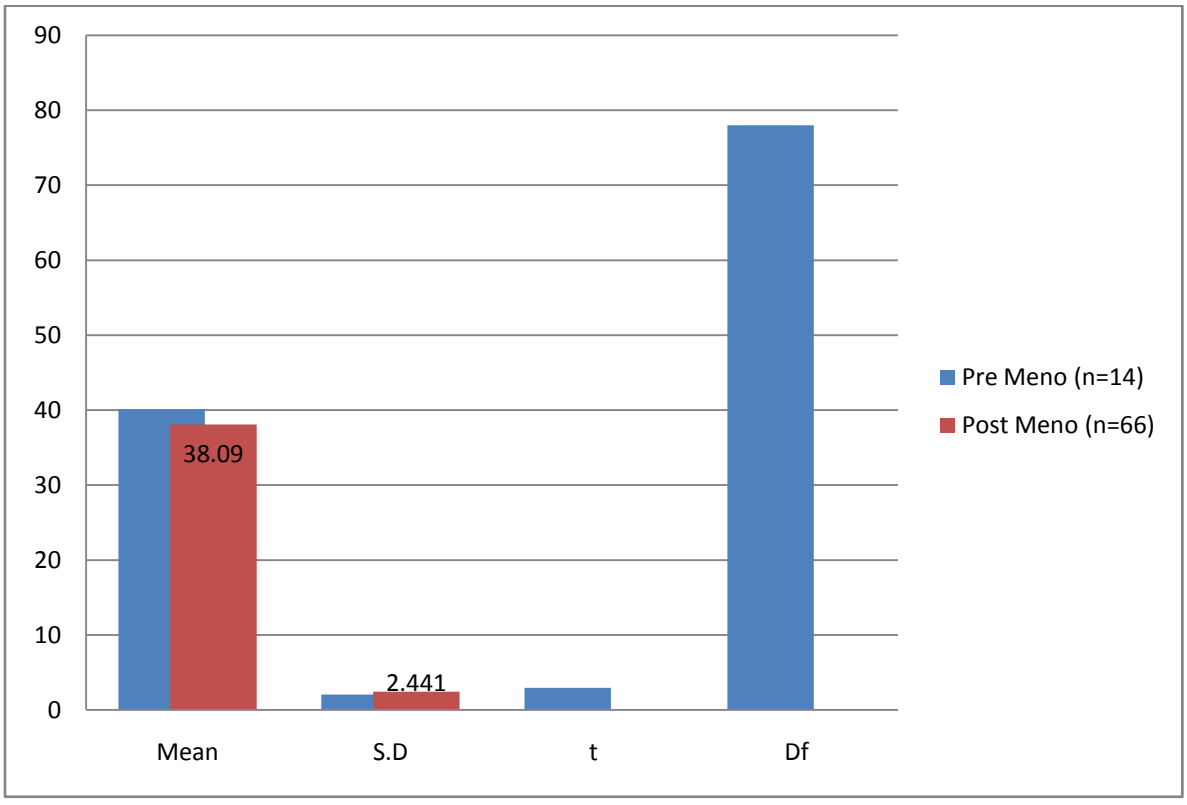
Graph



T-Test

HDL	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	40.14	2.033	2.932	78	.004<0.05
Post Meno (n=66)	38.09	2.441			Significant

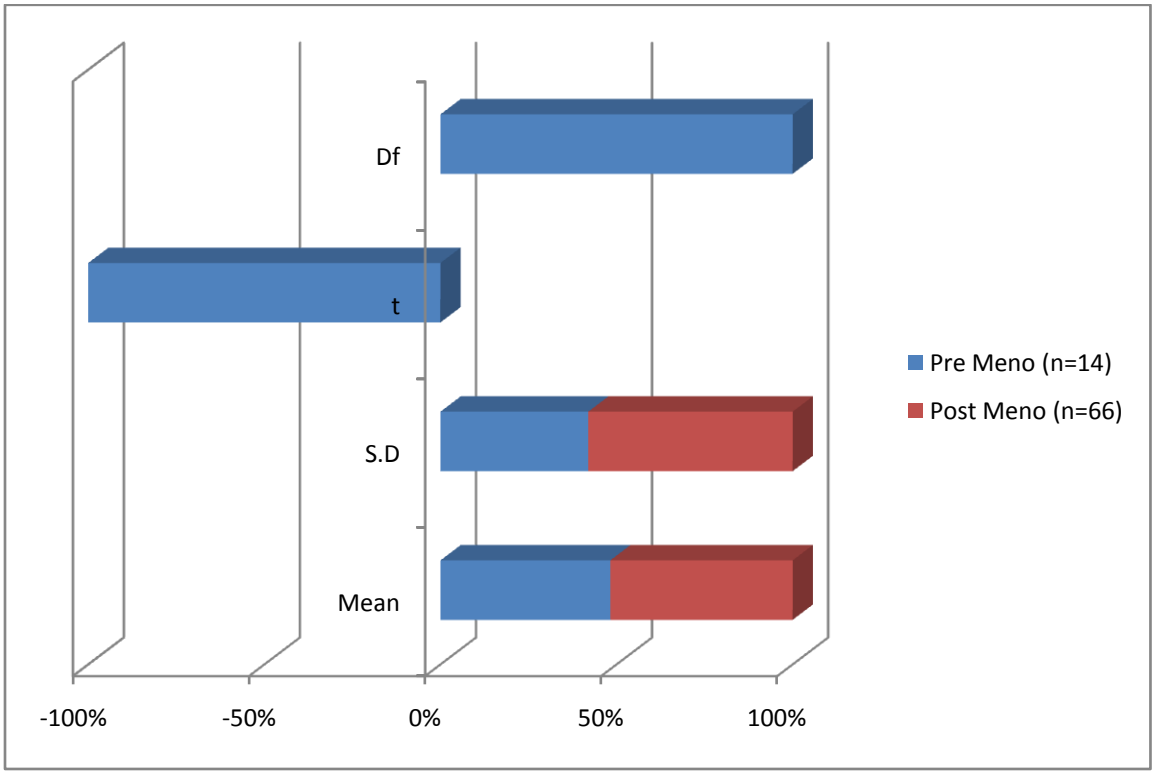
Graph



T-Test

LDL	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	106.06	18.607	-1.081	78	.283>0.05
Post Meno (n=66)	113.91	25.739			Not Significant

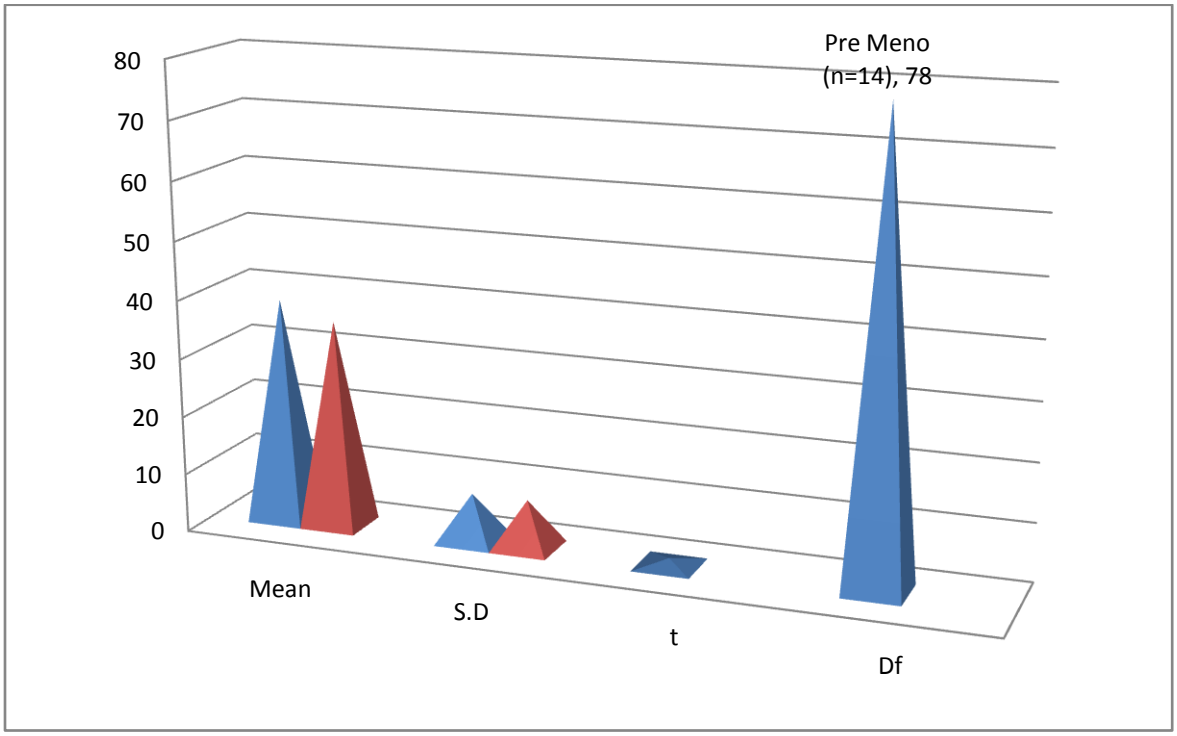
Graph



T-Test

VLDL	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	38.37	8.147	1.206	78	.231>0.05
Post Meno (n=66)	35.48	8.152			Not Significant

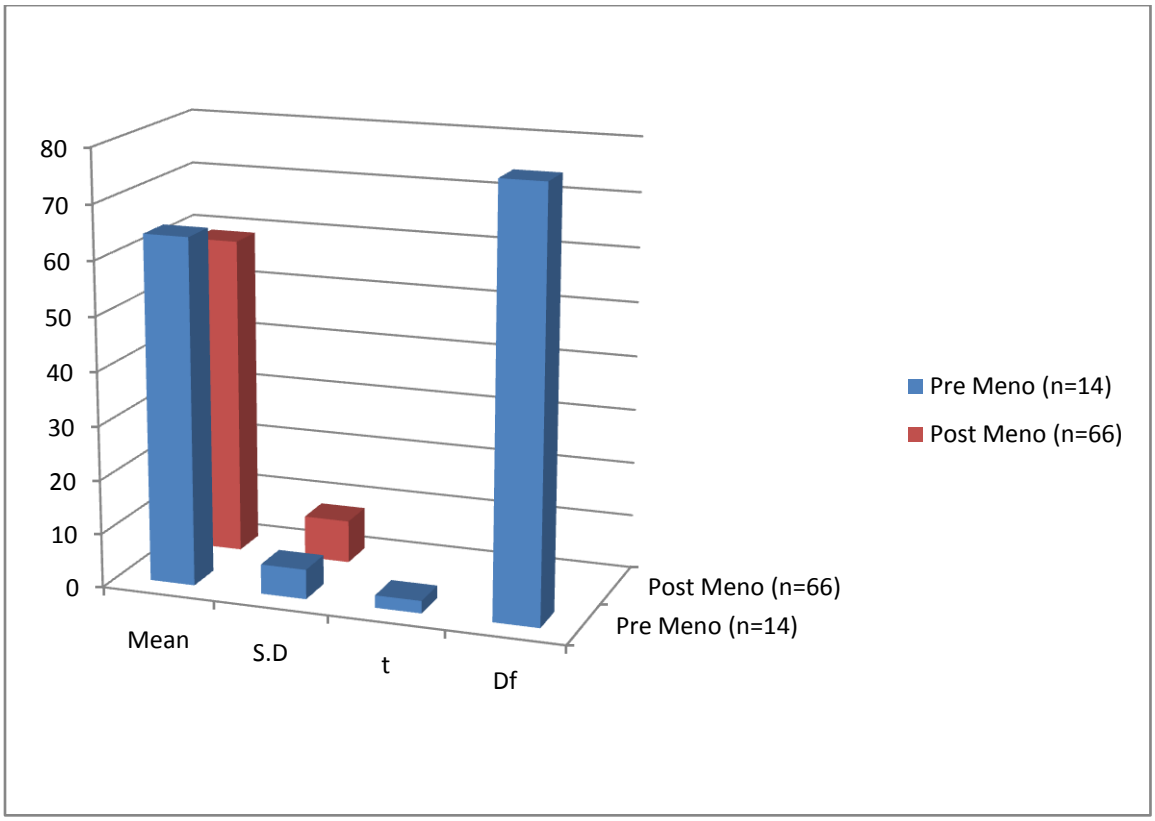
Graph



T-Test

EF	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	64.00	5.491	2.309	78	.024<0.05
Post Meno (n=66)	58.88	7.883			Significant

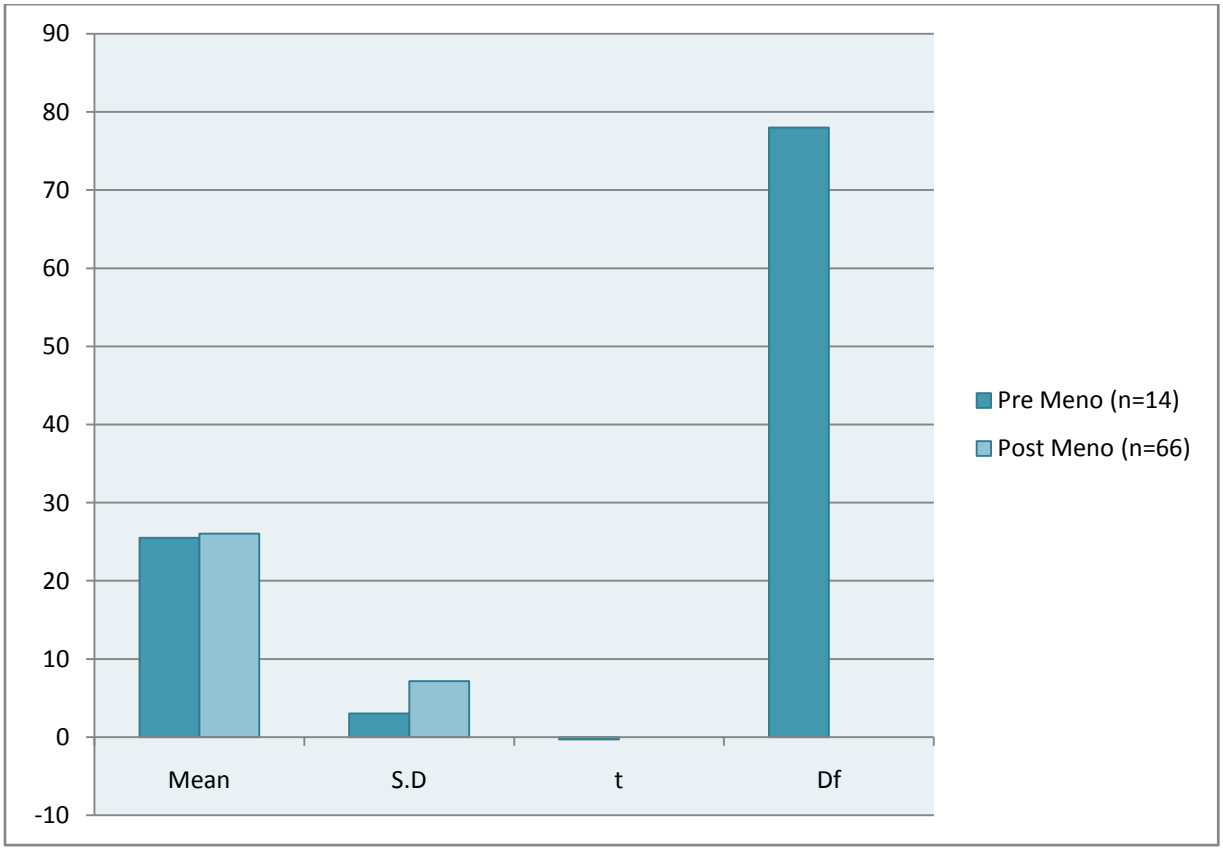
Graph



T-Test

Bld.urea	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	25.50	3.032	-.279	78	.781>0.05
Post Meno (n=66)	26.05	7.146			Not Significant

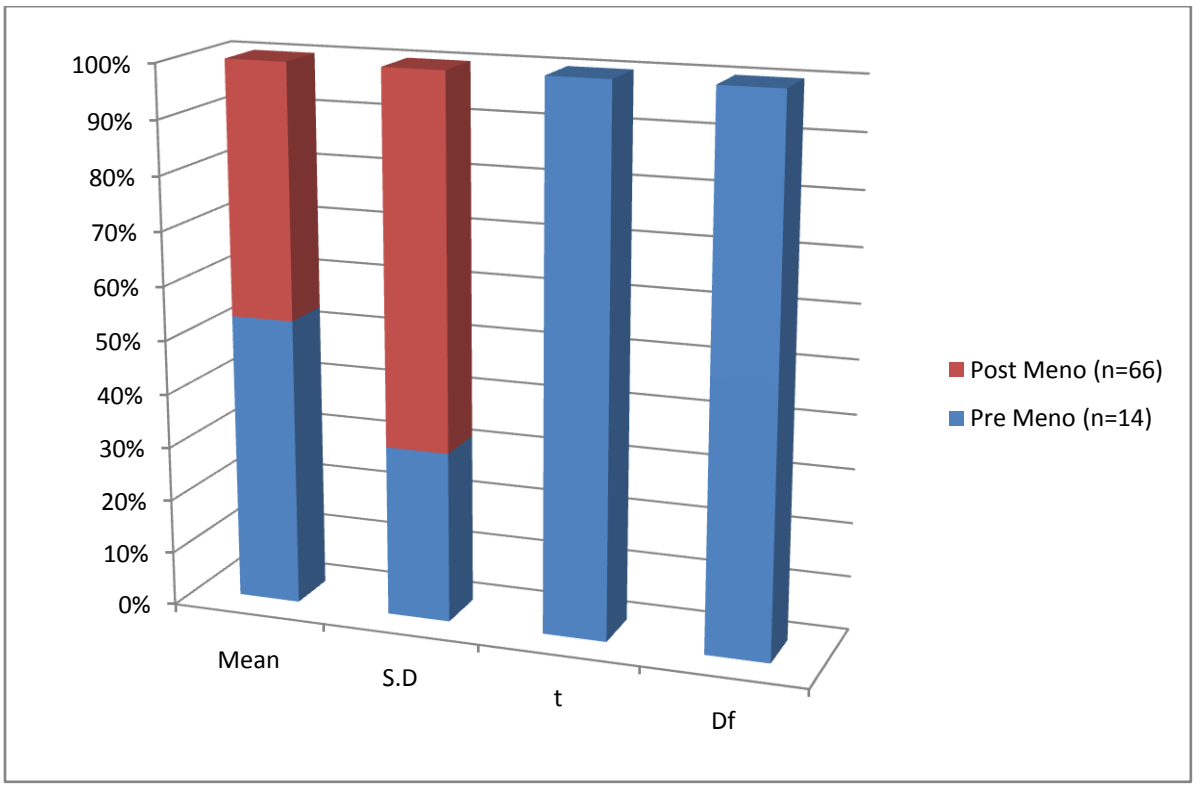
Graph



T-Test

S.Creat	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	1.1000	.12403	1.855	78	.067>0.05
Post Meno (n=66)	.9636	.26798			Not Significant

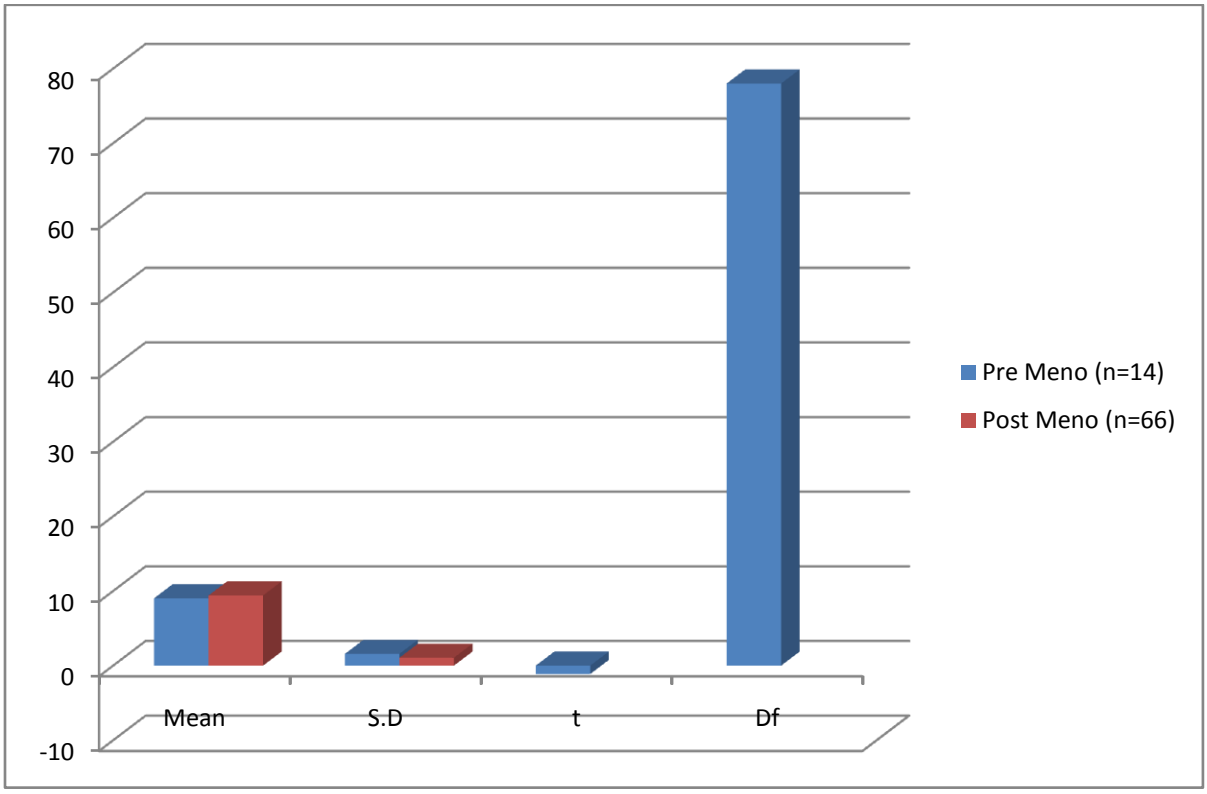
Graph



T-Test

HB	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	9.0214	1.57196	-1.120	78	.266>0.05
Post Meno (n=66)	9.4030	1.05568			Not Significant

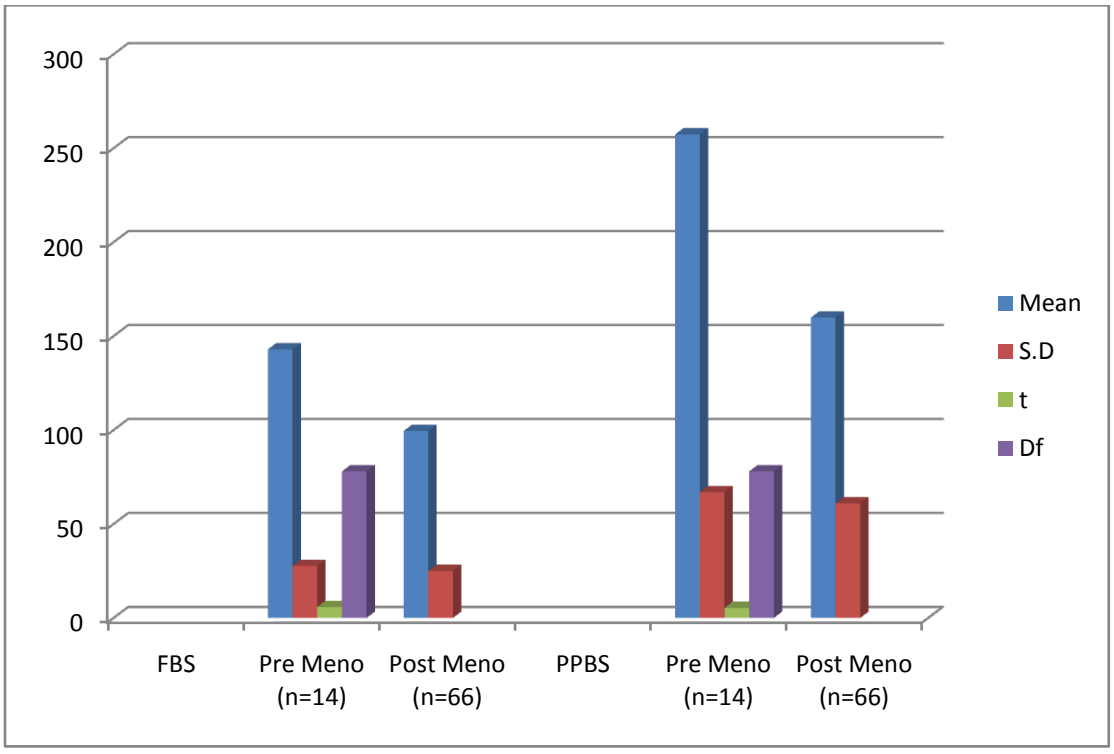
Graph



T-Test

	Mean	S.D	t	Df	Statistical inference
FBS					
Pre Meno (n=14)	142.93	27.647	5.795	78	.000<0.05
Post Meno (n=66)	99.53	24.990			Significant
PPBS					
Pre Meno (n=14)	257.36	66.890	5.345	78	.000<0.05
Post Meno (n=66)	159.85	60.980			Significant

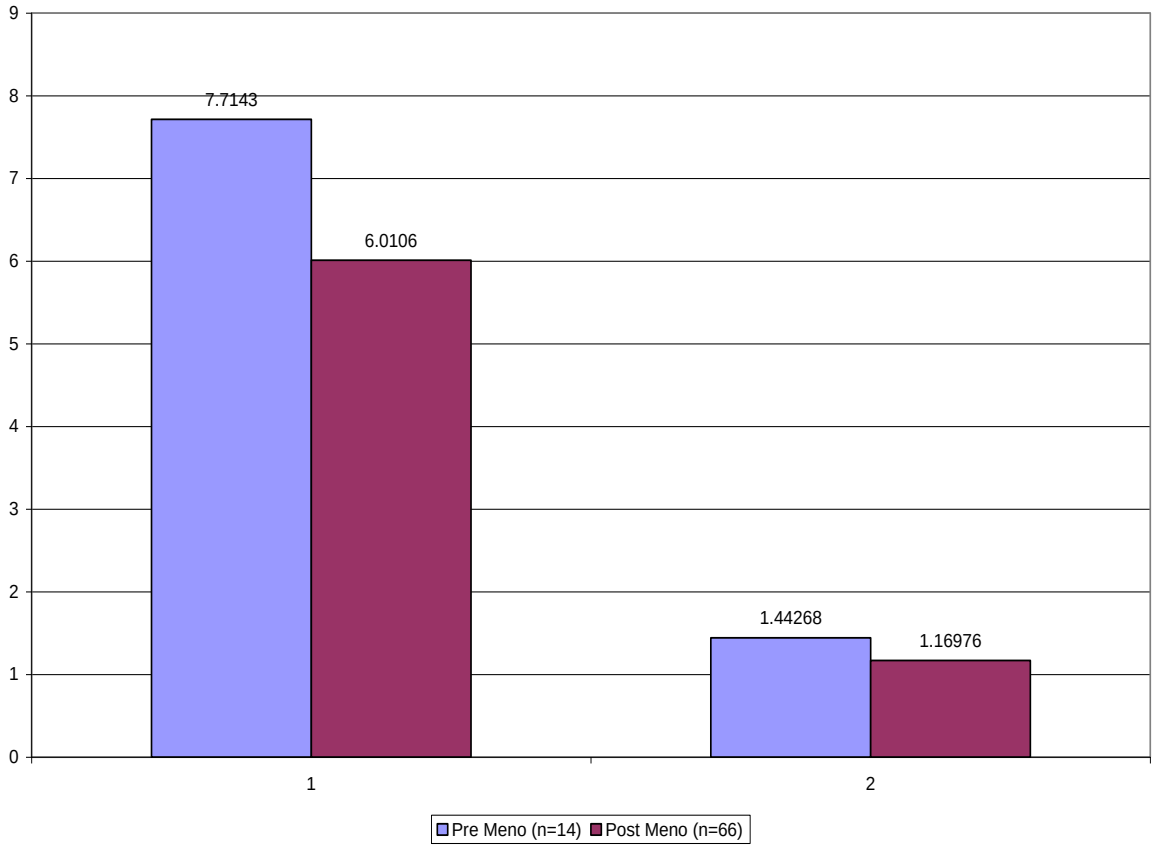
Graph



T-Test

Hb1AC	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	7.7143	1.44268	4.748	78	.000<0.05
Post Meno (n=66)	6.0106	1.16976			Significant

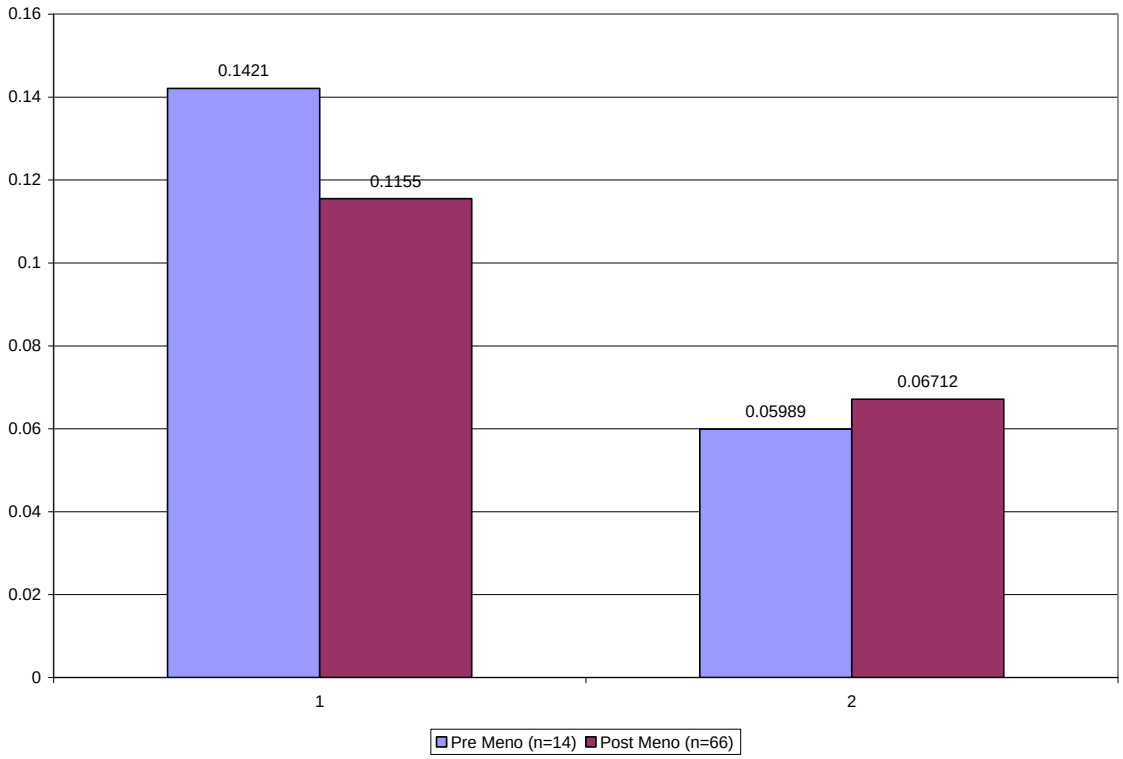
Graph



T-Test

Urine PCR	Mean	S.D	t	Df	Statistical inference
Pre Meno (n=14)	.1421	.05989	1.375	78	.173>0.05
Post Meno (n=66)	.1155	.06712			Not Significant

Graph



DISCUSSION

DISCUSSION

This study was conducted to find out risk factors and clinical features of coronary artery disease in women. The study population was 80 patients. They are divided into premenopausal and postmenopausal. 14 patients were in premenopausal group and 66 patients were postmenopausal group. Fasting lipid profile was measured using an autoanalyser after 12 hrs of fasting. FBS, PPBS, Hb1AC, Urine PCR, BMI were also calculated. The results were statistically analyzed.

Age distribution :

The mean age for premenopausal group was 40 years. The young MI that involving in CAD is 38 years. CAD occurs between 46 to 59 years of age.

Distribution of BMI among the groups:

Mean BMI among premenopausal age was 26, S.D 4.79. ie., They are overweight. BMI among postmenopausal age was 23. S.D.2.671. We compared BMI between Pre and Postmenopausal age groups; the p value is significant (p 0.001), obesity plays significant role for causation of CAD among women.

Mean total cholesterol in premenopausal women is 184.57 SD 22.3. The mean total cholesterol among postmenopausal women 187.02, S.D

25.049. On statistical analysis we found that there is no significant alteration in cholesterol between premenopausal and postmenopausal women.

TGL :

We observed that TGL were significantly elevated in premenopausal women (mean 191.86) S.D 40.733. For postmenopausal women mean was 177.39 with S.D of 40.76. There is no statistical significance between two groups.

HDL :

In our study the mean HDL among premenopausal women is 40.14, S.D 2.033, and among postmenopausal women 38.09, SD 2.441. This was found very significant on statistical analysis with a p value < 0.004.

LDL :

In our study mean LDL among premenopausal women was 106.06, S.D 18.607 and among postmenopausal women it was 113.91, S.D 25.739. We didn't find any statistically significant difference in LDL level between premenopausal and postmenopausal women.

VLDL :

We observed that, there is no significant rise in serum VLDL level between pre and postmenopausal women (p value 0.23, 70.05). The mean

value of VLDL among premenopausal women was 38.37; S.D 8.147 and among postmenopausal women was 35.48, S.D 8.152.

BP :

There is no significant statistical difference between premenopausal and postmenopausal groups. We observed that 37.5 % of women having high BP and 62.5 % have normal BP.

FBS & PPBS :

FBS in premenopausal in an average was 142.93; S.D 27.647 and postmenopausal women it was 99.53, SD 24.99. There is significant statistical difference between premenopausal and postmenopausal women in FBS value. PPBS value also got significant difference between pre and postmenopausal women.

PPBS in premenopausal women was 257.36 S.D 66.89 and postmenopausal women it was 159.82, S.D 60.98.

Hb_1AC :

We observes that Hb_1AC were significantly elevated among premenopausal women 7.7143, S.D 1.442 and among postmenopausal women it was 6.01, S.D 1.69. This was found very significant on statistical analysis with a p value of <0.001

ECG :

ST segment elevation MI was observed in 33% of patients. 16% were NSTEMI and 31 % were unstable angina. Most of the patient present late to the hospital. In ECG Q was present in most of the patient present in evolved stage and most of them were lacking typical symptoms.

ECHO :

On ECHO Regional wall motion abnormalities were present in 30 % of patients and it was absent in 50 % of patients. Some present early in hyperacute phase itself . So myocardial salvage was good.

EF :

We observed statistically significant difference in ejection fraction between premenopausal and postmenopausal women (p 0.024 < 0.05). The average ejection fraction among premenopausal women was 64.00 S.D 5.491, and among postmenopausal women was 58.88, S.D 7.883. Women with CAD present late to the hospital.

CONCLUSION

CONCLUSION

1. The presentation of CAD in young women is increasing in our study.
2. Occupational stress, family stress and psychosocial factors are important risk factors in young MI.
3. Obesity and metabolic syndrome including (PCOS) polycystic ovarian syndrome are noted as risk factors in our study.
4. The anginal symptoms of CAD are lacking in women with diabetes . As typical symptoms of CAD are lacking, they presented in evolved stage of MI.
5. Systemic hypertension and diabetes increases CAD mortality and morbidity.
6. In our study low HDL and high LDL are noted as CAD risk factors.
7. Early menopause in women had contributed to the development of CAD.
8. Congestive cardiac failure was common in those women who presented late to the hospital.

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BIBLIOGRAPHY

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APPENDIX

MASTER CHART

Case	Sample	Age	weight	Height	BMI	BP	FBS	PPBS	Hb1AC	TC	TGL	HDL	LDL	VLDL	Urine PCR	ECG	ECHO	EF	Bld. urea	S.Crea	HB
1	2	48	70	160	27.34	120/70	72	88	5.50	180	162	38	110	32	0.15	stemi	RWMA+	55	42	.60	7.80
2	1	39	69	156	28.35	130/90	161	270	7.20	210	158	42	136	32	0.10	IWMI	RWMA+	58	21	1.10	9.20
3	2	54	62	165	22.77	140/100	138	250	8.00	200	140	36	136	28	0.20	NSTEMI	RWMA -	63	20	1.20	10.70
4	2	48	65	172	21.97	130/80	76	135	5.60	210	180	35	139	36	0.14	NSTEMI	RWMA -	65	28	.90	9.80
5	2	56	65	150	28.89	110/70	88	92	4.90	222	172	34	154	34	0.10	stemi	RWMA+	59	34	.80	11.20
6	2	52	65	165	23.88	120/80	130	170	7.40	178	160	30	116	32	0.02	NSTEMI	RWMA+	66	29	1.30	10.00
7	2	46	75	151	32.89	130/100	130	250	8.40	160	170	34	92	34	0.16	stemi	RWMA -	67	25	.70	9.00
8	2	52	68	170	23.53	110/80	78	119	5.60	178	172	39	105	34	0.13	Ischaemia	RWMA -	69	33	.90	8.20
9	2	49	60	161	23.15	120/70	118	170	5.80	160	106	38	101	21	0.02	NSTEMI	RWMA -	61	31	1.20	9.90
10	1	42	68	153	29.05	110/90	128	318	8.00	172	170	42	96	34	0.14	STEMI	RWMA+	63	24	1.10	8.80
11	2	47	59	162	22.48	160/90	106	160	5.90	168	119	36	108	24	0.30	Ischaemia	RWMA -	62	26	.80	10.00
12	2	65	66	170	22.84	140/90	78	118	5.70	170	250	36	84	50	0.14	stemi	RWMA -	56	30	.70	10.50
13	2	52	66	165	24.24	110/80	88	115	4.60	170	118	40	106	24	0.11	Ischaemia	RWMA -	50	35	.90	11.00
14	2	48	60	154	25.30	150/90	138	275	8.40	180	310	36	82	62	0.02	Ischaemia	RWMA -	57	22	1.20	11.50
15	1	39	73	160	28.52	120/70	160	262	9.20	160	158	38	90	32	0.16	STEMI IW	RWMA -	68	27	1.30	7.90
16	1	42	57	159	22.55	160/100	120	165	8.40	210	250	42	118	50	0.15	NSTEMI	RWMA -	69	28	1.10	8.20
17	2	64	52	162	19.81	170/100	80	110	5.60	190	310	35	93	62	0.18	stemi	RWMA+	54	18	1.00	9.30
18	2	58	62	170	21.45	130/80	78	140	5.70	178	160	36	110	32	0.10	Ischaemia	RWMA -	68	15	.90	10.10
19	2	75	68	170	23.53	140/90	102	190	5.20	220	170	41	145	34	0.12	Ischaemia	RWMA -	66	21	.60	9.80
20	2	70	59	159	23.34	110/70	128	210	7.00	186	180	35	115	36	0.15	NSTEMI	RWMA -	53	31	.70	8.60
21	1	41	61	152	26.40	120/80	130	240	7.60	178	168	38	106	34	0.14	STEMI	RWMA+	57	23	.90	9.70
22	1	45	66	180	20.37	130/90	128	128	4.90	158	192	37	83	38	0.17	stemi	RWMA -	69	21	1.10	10.00
23	1	38	64	149	28.83	140/80	200	380	8.60	164	224	41	78	45	0.06	STEMI	RWMA+	66	28	1.20	11.00
24	2	47	56	152	24.24	130/90	88	170	5.60	198	250	42	106	50	0.18	Ischaemia	RWMA -	62	19	.60	11.50
25	2	54	68	164	25.28	160/100	78	110	4.90	210	110	38	150	22	0.11	stemi	RWMA+	55	15	.90	9.00
26	2	58	60	166	21.77	180/110	96	120	5.00	200	186	39	124	37	0.07	stemi	RWMA+	59	21	1.40	9.30
27	2	53	64	164	23.80	160/100	131	250	7.40	160	170	40	86	34	0.08	Ischaemia	RWMA -	70	30	.80	8.50
28	2	49	70	167	25.10	170/100	140	240	8.50	210	250	39	121	50	0.11	STEMI	RWMA+	57	18	1.10	6.80
29	2	56	67	170	23.18	140/90	157	210	7.40	194	220	40	110	44	0.18	NSTEMI	RWMA -	58	21	1.00	9.10
30	2	47	48	154	20.24	110/70	76	110	5.40	178	160	36	110	32	0.11	Ischaemia	RWMA -	65	23	.60	8.60
31	2	52	64	170	22.15	130/80	78	140	5.50	190	175	38	117	35	0.08	Ischaemia	RWMA -	63	14	1.50	11.00
32	2	58	65	160	25.39	140/90	140	255	7.20	174	209	39	93	42	0.06	stemi	RWMA+	47	17	1.10	10.60
33	2	64	61	156	25.07	130/80	78	111	5.60	180	176	38	107	35	0.09	Ischaemia	RWMA -	54	25	1.00	9.40
34	2	62	67	172	22.65	140/90	76	98	4.60	222	158	36	154	32	0.08	NSTEMI	RWMA -	52	22	.90	8.70
35	2	68	63	157	25.56	170/100	89	110	4.90	194	173	38	121	35	0.10	stemi	RWMA+	42	23	.75	9.50
36	2	72	70	176	22.60	160/90	77	113	5.30	180	172	35	111	34	0.03	Ischaemia	RWMA -	57	25	.90	10.00
37	2	66	69	165	25.34	140/80	150	292	8.40	192	160	37	123	32	0.05	stemi	RWMA+	45	30	.80	10.50
38	2	48	70	160	27.34	120/70	76	114	5.90	247	198	38	169	40	0.11	NSTEMI	RWMA -	58	32	1.60	9.70
39	2	72	64	170	22.15	130/80	68	127	5.90	190	179	40	114	36	0.08	Ischaemia	RWMA -	60	41	.80	8.60
40	2	52	65	169	22.76	110/70	142	265	8.50	175	169	39	102	34	0.09	STEMI	RWMA+	64	37	.75	8.40
41	2	67	52	162	19.81	150/90	92	125	5.60	173	201	37	96	40	0.17	Ischaemia	RWMA -	67	30	.90	8.00
42	2	75	58	157	23.53	160/100	136	239	7.40	175	168	38	103	34	0.06	stemi	RWMA+	42	25	1.00	9.50

Case	Sanple	Age	weight	Height	BMI	BP	FBS	PPBS	HbıAC	TC	TGL	HDL	LDL	VLDL	Urine PCR	ECG	ECHO	EF	Bld. urea	S.Crea	HB
43	1	39	80	150	35.56	150/80	172	325	9.20	184	236	41	96	47	0.17	steni	RWMA+	57	28	1.10	6.00
44	1	44	75	165	27.55	140/90	180	264	9.50	178	159	42	104	32	0.12	stemi	RWMA+	65	29	1.30	7.50
45	2	55	57	154	24.03	130/80	69	86	5.60	115	252	37	28	50	0.16	Ischaemia	RWMA -	69	23	1.60	8.20
46	2	46	69	160	26.95	120/80	139	261	8.50	119	179	34	49	36	0.03	stemi	RWMA -	54	21	1.10	9.30
47	2	52	65	168	23.03	130/90	68	110	5.70	178	116	39	116	23	0.05	NSTEMI	RWMA -	58	18	1.00	9.50
48	2	54	46	158	18.43	150/80	78	88	5.60	188	178	38	114	36	0.06	Ischaemia	RWMA -	66	18	.90	8.60
49	2	47	65	170	22.49	160/100	72	86	5.50	192	160	41	119	32	0.10	Ischaemia	RWMA -	67	25	.60	10.00
50	1	39	48	154	20.24	170/120	110	198	5.60	165	152	43	92	30	0.30	Ischaemia	RWMA -	70	24	.90	11.10
51	2	75	49	153	20.93	110/80	76	86	5.20	172	170	40	129	34	0.18	NSTEMI	RWMA -	55	21	.80	10.00
52	2	69	70	165	25.71	160/100	122	265	7.00	168	174	39	94	35	0.10	stemi	RWMA+	46	19	1.10	9.50
53	2	80	65	170	22.49	150/80	78	110	5.20	159	178	38	85	36	0.15	Ischaemia	RWMA -	58	24	1.30	9.90
54	2	46	65	164	24.17	160/90	110	225	6.40	238	192	41	159	38	0.02	stemi	RWMA+	57	39	1.50	8.70
55	2	47	60	152	25.97	140/100	119	235	6.50	252	210	39	171	42	0.17	stemi	RWMA+	54	40	1.40	7.80
56	2	52	55	153	23.50	130/80	78	111	5.80	196	116	38	135	23	0.18	Ischaemia	RWMA -	68	41	1.10	8.70
57	2	56	54	164	20.08	140/90	96	142	5.40	178	132	35	117	26	0.06	Ischaemia	RWMA -	59	25	.90	7.60
58	2	56	60	153	25.63	160/90	121	198	5.90	188	160	39	117	32	0.05	stemi	RWMA+	49	27	.80	8.50
59	2	65	55	168	19.49	130/80	86	126	5.60	178	176	37	106	35	0.10	NSTEMI	RWMA -	58	28	.70	8.40
60	2	69	70	175	22.86	110/80	78	138	4.90	161	178	42	83	36	0.16	Ischaemia	RWMA -	56	18	.60	9.30
61	2	72	62	154	26.14	120/70	110	221	4.70	210	199	44	126	40	0.05	stemi	RWMA+	43	20	.90	10.10
62	1	38	55	169	19.26	120/70	138	248	8.30	201	210	41	118	42	0.08	Ischaemia	RWMA -	67	24	1.10	11.50
63	1	40	70	173	23.39	140/100	128	296	8.60	234	278	39	139	56	0.20	NSTEMI	RWMA -	68	25	1.00	7.80
64	2	49	64	160	25.00	170/100	130	259	9.40	261	164	38	190	33	0.30	NSTEMI	RWMA -	70	30	1.20	9.60
65	2	69	68	168	24.09	140/80	66	110	5.60	198	176	37	126	35	0.40	Ischaemia	RWMA -	69	28	.90	8.90
66	2	59	70	175	22.86	130/90	77	126	5.60	172	169	41	97	34	0.07	Ischaemia	RWMA -	66	29	.70	10.50
67	2	62	64	155	26.64	160/110	98	210	5.90	199	280	38	105	56	0.08	steni	RWMA+	48	32	.80	10.00
68	2	67	50	168	17.72	130/80	96	118	4.90	206	192	34	134	38	0.09	Ischaemia	RWMA -	61	30	1.10	11.30
69	1	41	70	153	29.90	160/100	140	299	7.20	178	160	38	108	32	0.10	STEMI	RWMA+	53	24	1.00	9.60
70	2	57	57	170	19.72	140/80	78	98	4.80	165	174	40	90	35	0.16	Ischaemia	RWMA -	66	38	1.40	9.70
71	2	78	70	165	25.71	150/100	110	214	6.00	196	161	39	125	32	0.11	STEMI	RWMA+	44	31	.60	8.20
72	2	65	58	165	21.30	140/80	78	98	5.50	178	168	39	105	34	0.12	NSTEMI	RWMA+	65	37	.70	8.50
73	1	44	74	152	32.03	120/70	106	210	5.70	192	171	38	120	34	0.10	STEMI	RWMA+	66	31	1.20	8.00
74	2	49	67	170	23.18	160/100	110	139	5.20	178	168	39	105	34	0.09	NSTEMI	RWMA -	70	18	1.30	9.00
75	2	72	57	166	20.69	130/80	100	140	5.30	182	169	41	107	34	0.10	Ischaemia	RWMA -	64	22	.60	9.50
76	2	65	69	170	23.88	160/110	121	210	6.10	170	161	38	100	32	0.12	STEMI	RWMA+	47	17	.90	6.80
77	2	58	49	168	17.36	110/70	100	128	4.50	191	148	42	119	30	0.15	Ischaemia	RWMA -	66	24	.80	9.90
78	2	48	56	170	19.38	120/80	102	126	4.70	193	150	40	123	30	0.11	Ischaemia	RWMA -	67	19	1.00	10.00
79	2	57	75	170	25.95	140/100	119	199	5.80	194	155	41	122	31	0.12	STEMI	RWMA+	50	21	1.20	11.00
80	2	52	68	175	22.20	130/80	88	96	5.60	176	140	40	108	28	0.10	Ischaemia	RWMA -	68	28	1.30	9.50

PROFORMA

APPENDIX

PROFORMA

Coronary Artery disease in women clinical profile and risk factors analysis

1. NAME :
2. AGE :
3. OP No / IP No :
4. ADDRESS :

5. OCCUPATION :
6. COMPLAINTS :

   History of presenting Complaints
7. PAST h/o :

   DM, HTN
8. PERSONAL h/o :

   Smoking, Alcohol
9. FAMILY h/o :
10. MENSTRUAL h/o :

11. OCCUPATIONAL h/o :

OCCUPATIONAL STRESS

12. DRUG h/o :

OCP pills user, hormone

replacement therapy

General Examination :

BP

PR

RR

TEMPREATURE

WEIGHT

HEIGHT

BMI

Systemic Examination :

CVS, RS, CNS

ABDOMEN

Investigations :

CBC

Hb

TC

DC

ESR

PCV

PLT

RBS

UREA

CREATININE

LIPID PROFILE

SGOT

SGPT

URINE PCR

ECG

ECHO

USG ABDOMEN

ABBREVIATIONS AND ACRONYMS

ABBREVATIONS

ACS	-	Acute Coronary Syndrome
AMI	-	Acute Myocardial Infarction
BP	-	Blood Pressure
CAD	-	Coronary Artery Disease
CPK	-	Creatine Phosphokinase
CPR	-	Cardiopulmonary Resuscitation
CV	-	Cardiovascular
DVT	-	Deep Vein Thrombosis
ECG	-	Electrocardiogram
EF	-	Ejection Fraction
FBS	-	Fasting Blood Sugar
Hb_1AC	-	Glycosylated Hemoglobin
HDL	-	High Density Lipoprotein
HRT	-	Hormone Replacement Therapy
ICD	-	Implantable Cardioverter Defibrillator
LA	-	Left Atrium
LAD	-	Left anterior descending
LCX	-	Left Circumflex
LDL	-	Low Density Lipoprotein

LV	-	Left Ventricle
MI	-	Myocardial Infarction
NSTEMI	-	Non ST Segment elevation Myocardial Infarction
PCI	-	Percutaneous coronary Intervention
Postmeno	-	Postmenopausal
PPBS	-	Post Prandial Blood Sugar
PR	-	Pulse Rate
Premeno	-	Premenopausal
RCA	-	Right Coronary artery
RR	-	Respiratory Rate
STEMI	-	ST Segment elevation Myocardial Infarction
TC	-	Total Cholesterol
TGL	-	Triglycerides
UA	-	Unstable Angina
VLDL	-	Very Low Density Lipoprotein

ETHICAL COMMITTEE APPROVAL

K.A.P.VISWANATHAM GOVT. MEDICAL COLLEGE
TIRUCHIRAPALLI -1
INSTITUTIONAL ETHICS COMMITTEE

CERTIFICATE OF CLEARANCE

This is to certify that the project work titled Study of coronary artery disease in women-clinical profile and risk factors proposed by Dr.T.Chakravarthi part of fulfillment of M.D/M.S course in the subject of Medicine for the year 2013-2016 by The Tamilnadu Dr.MGR Medical University has been cleared by the Ethics committee.

13/12/14

CHAIRMAN,
Institutional Ethics Committee
K.A.P.Viswanatham Govt. Medical
College, Tiruchirapalli -1

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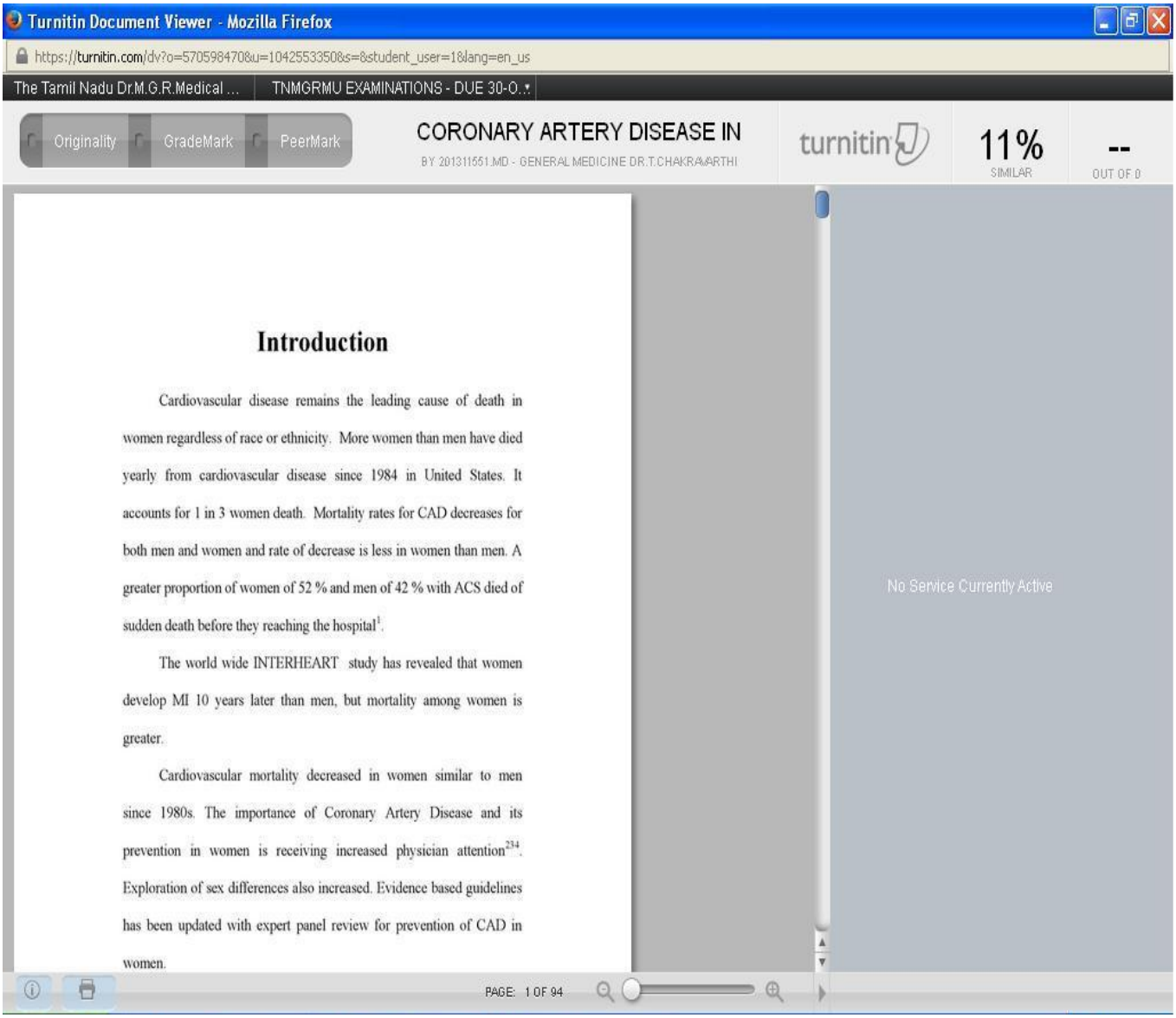
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Cardiovascular disease remains the leading cause of death in women regardless of race or ethnicity. More women than men have died yearly from cardiovascular disease since 1984 in United States. It accounts for 1 in 3 women death. Mortality rates for CAD decreases for both men and women and rate of decrease is less in women than men. A greater proportion of women of 52 % and men of 42 % with ACS died of sudden death before they reaching the hospital[1].
The world wide INTERHEART study has revealed that women develop MI 10 years later than men, but mortality among women is greater.
Cardiovascular mortality decreased in women similar to men since 1980s. The importance of Coronary Artery Disease and its prevention in women is receiving increased physician attention[234]. Exploration of sex differences also increased. Evidence based guidelines has been updated with expert panel review for prevention of CAD in women.
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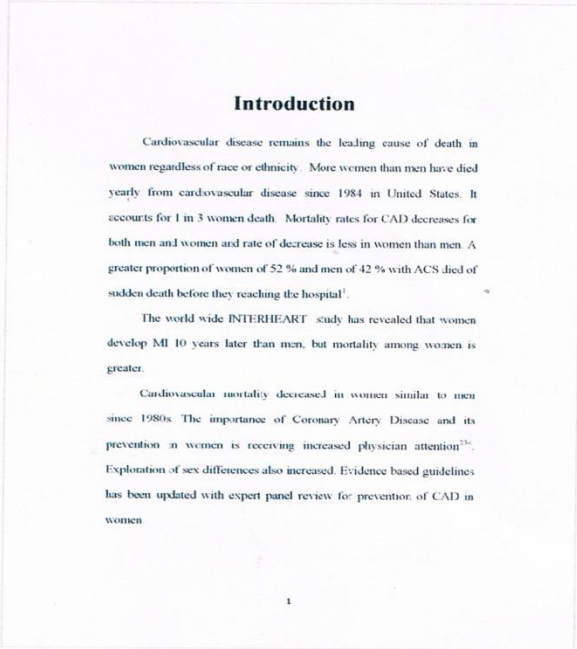

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